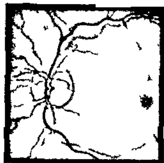
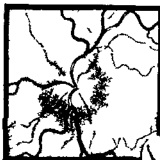
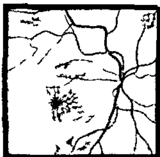
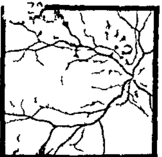




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THE FUNDUS OCULI

HUTCHISON'S

CLINICAL METHODS

by

DONALD HUNTER, C B E, M D, F R C P

Physician to the London Hospital

and

R R BOMFORD D M, F R C P

Physician to the London Hospital

THIRTEENTH EDITION



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PREFACE TO THE THIRTEENTH EDITION

With this edition Sir Robert Hutchison retires from active participation in a book which he wrote with the late Dr Harry Rainy and has since guided through twelve editions in fifty nine years To mark this event which must be almost unique in medical publishing we have altered the title of the thirteenth edition to *Hutchison's Clinical Methods* We have done this to assure Sir Robert, in the country fastness to which he has retired that what he has called his baby shall not die if we can help it and as long as it survives whatever names come and go that of Hutchison shall continue with it

The present edition is nearly twenty pages shorter than the last We are confident nevertheless that it contains a good account of those established methods of clinical investigation which are likely to be most useful to students and practitioners For more highly specialized methods and less well established ones larger works must be consulted We hope too that the book still embodies the wise outlook which Sir Robert Hutchison has always upheld and taught. In these days enormous developments in the field of diagnosis have put a strain upon the judgment of the practitioner Now more than ever before he mu t avoid thinking that the laboratory and X ray room can save him from taking a careful history and making a thorough clinical examination of his patient

We are indebted to many of our colleagues for their assistance Dr Ronald Henson has been responsible for a detailed revision of the chapter on neurological examination Mr Charles Keogh has rewritten the examination of the throat nose and ear and has himself drawn new illustrations for this section Dr Malcolm Towers has been mainly responsible for the revision of the chapter on the cardiovascular system and for an entirely new section on electrocardiography We are indebted to Dr William Evans for allowing us to reproduce electrocardiograms taken in the Cardiac Department of the London Hospital We are also grateful to Dr Kenneth Tallerman (examination of children) Mr J E M Ayoub (examination of the eyes) Dr J S Pegum (examination of the skin) and Dr John Glover (respiratory system) for advice

on the subjects indicated One of us is particularly indebted to his late house physician Dr Geoffrey Mathews for valuable help in the whole of the revision

We also extend our thanks to Sir Gordon Holmes and to Messrs E & S Livingstone for permission to reproduce Figs 57–9 and 60 from *Introduction to Clinical Neurology* to Messrs Lea & Febiger and Henry Kimpton for Figs 39–41 from Burch s *A Primer of Cardiology* and to Dr Peter Boulden for further original drawings Sources of other new illustrations are acknowledged in the text

D H
R R B

PREFACE TO THE ORIGINAL EDITION

The title *Clinical Methods* probably describes the scope of this book better than any other It is not intended as a treatise upon medical diagnosis On that subject there is already a sufficiency of good works in existence It aims rather at describing those methods of clinical investigation by the proper application of which a correct diagnosis can alone be arrived at To every student when he first begins work in a medical ward the question presents itself How shall I investigate this case? To that question the present work is intended to provide an answer The first chapter deals therefore with the methods of case taking in general and includes a general scheme for the investigation of medical cases The rest of the book is really an expansion of that scheme each system being taken up separately and the methods of investigating it described in detail

A special chapter has been devoted to the clinical methods of examining children as these differ in many respects from those employed in the case of adults Chapters have also been added on the examination of Pathological Fluids and on Clinical Bacteriology subjects which are daily growing in importance The methods employed in the investigation of surgical gynæcological or obstetric cases do not fall within the scope of the work

No effort has been spared to make the book thoroughly up to date and it is hoped therefore that it will be found useful by those practitioners who may wish to make themselves acquainted with the latest methods of clinical investigation While the whole book has passed through the hands of both of us yet each has made himself specially responsible for certain parts Thus Dr Rainy has written Chapters II IV VI and XIV the sections on the electrical examination of muscle and nerves on the parasites of the alimentary tract and on the microscopical examination of the urine The rest of the work is from the pen of Dr Hutchison

In order to avoid burdening the text but few references have been given to authorities and original source We should like however to take this opportunity of acknowledging the help which we have received from various friends Amongst these are Drs

Alex Bruce R W Philip G Lovell Gulland and John Thomson who have helped us with criticism and advice in the preparation of Chapters IV and IX VI V and XII respectively We have also to thank Dr Patrick Manson Dr Byrom Bramwell Dr J Purves Stewart and Prof Symington for the use of specimens and illustrations and Dr T F Milroy for assistance in the revision of proofs To Dr R J M Buchanan we are specially indebted for preparing the drawings illustrating the microscopical examination of the blood

R H
H R

September 1897

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CHAPTER I

CASE-TAKING

THERE can be no question of the value of accurate and systematic case taking It trains the beginner in habits of thoroughness and exactness at the bedside and ensures that no point of importance in a case is missed About the *method* to be pursued in taking a case there is much difference of opinion and almost every clinical teacher has his own special plan And it is not of much importance what particular method one adopts provided one adheres to it Every good method of case taking should be both comprehensive and concise It should be comprehensive so as to be capable of being applied to every case and of covering all the points in it it should be concise so as to present all the important features of a case in as small a compass as possible The question of conciseness *is of great importance Nothing is more annoying than to be* obliged to wade through a mass of verbiage in order to get at the chief facts of a case The student should practise the art of presenting the leading features of a case in a few sentences and the writing of resumes of cases will be found a useful exercise He should avoid lengthy verbal descriptions Simple outline drawings can often convey information that would otherwise require many sentences

There is appended to this chapter a scheme of case taking which meets all ordinary requirements At the same time it must be used with judgment and elasticity All the points mentioned need not be inquired into in each individual case For example if a patient is suffering from advanced cardiac disease, there is no use in writing a minute description of the state of his teeth This often is the kind of error into which beginners fall Some experience is required to assess what special inquiries are important in particular cases and mistakes often happen at first but the application of common sense will ensure the avoidance of gross blunders

The taking of a case consists of two parts—

I The interrogation of the patient

II The physical examination

Clinical clerks will find it best to make rough notes of both of these and afterwards to write out the whole case in detail

I THE INTERROGATION OF THE PATIENT

The object of interrogation is to elicit information regarding *the patient s present illness the state of his previous health and that* of his family The interrogation must be patiently carried out the patient being allowed as far as possible to tell his story in his own words One patient is a good witness and another poor One gives an excellent history Another has to have the history of *his illness dragged out of him by methods of slow extortion and* even then a great deal of what he says may prove irrelevant Some patients seem quite unable to give any precise account of what they feel to be wrong This may be due to stupidity or to the effects of disease on their mental faculties Some however fail to appreciate the need for accurate information and feel that if only they can impress the doctor with the urgency of their distress (which in such cases often turns out to be emotional in nature) he will be able as if by magic to relieve them of it It is important to recognize the reason for the evasiveness of such patients and not to allow oneself to become annoyed with them The manner of questioning may make all the difference in the world to a nervous or suspicious patient One may defeat one s own ends by wounding the senti ments or conscience of the patient long before the physical examina tion begins It is also important to avoid asking the same question twice because this conveys to the patient the impression of taking but a languid interest in his case

All symptoms are not of equal diagnostic importance There is usually one symptom which troubles the patient more than any other and which can be described as the *presenting symptom* Special attention should always be given to it It is a good rule not to ask leading questions when taking the history but once a tentative diagnosis has been made on the presenting symptom it is well to ask for corroborating symptoms which the patient may not have specially observed or which he may have omitted to mention The experienced doctor shows great skill in the choice and wording *of his leading questions The methodical student will learn this* art by experience

1 The general interrogation —Begin by ascertaining the patient s name age occupation and whether he is married or single It is also important to note his exact postal address

Two important questions then follow—(1) Of what does he complain ? It is a mistake to ask What is the matter ? as this lays one open to the retort that that is what the patient came to find out (2) How long have the symptoms been present ? The patient usually dates the start of his illness from some impressive event or from the onset of some severe symptom Questioning will sometimes reveal earlier symptoms which belong to the history of the present illness The real date of onset can often be more accurately defined by asking such questions as When were you last quite well ? Sometimes it is useful to ask Did you ever have a pain like this before ? Having thus defined his com plaint and its duration proceed to ascertain the chief facts in his history

The most logical plan is to take the family history first, but in practice it is perhaps more convenient to begin with the history of the present illness to pass from that to an inquiry into the patient s previous health and thence to the family history It is usually sufficient to inquire regarding the state of health or cause of death of the immediate relatives only—the parents brothers and sisters and if the patient is married of his own children if any These facts may tell us whether he is predisposed by heredity to any particular disease or whether he has reason to be anxious about a particular disease because he has seen a relative suffer and per haps die from it

Next comes the social history which includes the patient s mental attitude to his life and his work Here it is well to begin with what may be grouped together as the patient s physical and emotional environment his surroundings both at home and at work, and his habits One should endeavour to visualize the life of one s patient, sharing his emotions and viewing step by step his daily habits his diet and his work. It will often be found advantageous to ask the patient to give a brief account of a typical day

Inquire into (*a*) the exact nature of his occupation (not merely the name of his trade but what precisely his work involves) and whether or not it exposes him to injurious influences former occupations should also be noted Sometimes one should inquire into a patient s business affairs his ambitions anxieties quarrels

and in general his attitude to his work (*b*) His home surroundings their sanitary conditions and the possible existence of overcrowding (*c*) His domestic relationships his feelings about the other members of his family his own psychological make up his interests his hobbies his hopes his fears the holidays he gets and whether he enjoys them the amount of exercise he takes the games he plays and in general the sort of life he leads and the sort of person he is (*d*) The nature of his food and the extent of his indulgence in such articles as alcohol and tobacco (*e*) Whether or not he has lived abroad and if so in what part of the world and whether he was ill there

One should take up next the question of the patient s previous health Ascertain what illnesses he has had when he had them and their duration Be careful not to accept uncritically a diagnosis of a previous illness find out by whom the diagnosis was made and confirm this if possible by a few questions as to the symptoms experienced In all cases the patient should be questioned specifically for a history of rheumatic fever chorea scarlet fever tonsillitis pneumonia and exposure to tropical disease Adult males should be asked whether they have had a venereal disease and if necessary whether they have run the risk of it In the case of female patients information regarding venereal disease should as far as possible be obtained indirectly and in these circumstances questions should be put as delicately as possible Female patients should also be asked about menstruation In the majority of cases menstruation occurs every 28 days but the intervals may be longer or shorter according to the patient s habit If menstruation has ceased one must inquire how long it has been absent Normally the cessation of menstruation or menopause should not occur until about the 45th year or later It is also necessary to inquire whether the patient is losing more or less blood than usual The menstrual flow is to be regarded as abnormal if it lasts for less than two or more than eight days The presence or absence of pain at the periods must also be noted

We are now ready to obtain the history of the present disorder Ask how and when it began getting dates of the chief events if possible and whether suddenly or gradually what was the first thing the patient noticed wrong what has been the order of appearance of his symptoms and which are those that chiefly trouble him at the present time The use of medical or pseudo

medical terms should be gently but firmly discouraged A patient, for instance who says his complaint is catarrh or rheumatism should be asked to describe what exactly it is that he feels wrong Ascertain whether or not he has already been under treatment and if so what has been done for him

This completes the general interrogation and includes the chief facts that have to be inquired into in every case

2 The special interrogation must be modified according to the particular organ which is thought to be affected and the nature of the disease of which it is suspected to be the seat It is only by experience that one can tell what it is essential to ask in each individual case In order to help the beginner there follows a scheme of interrogation which he can pursue when he has reason to suppose that the patient s general symptoms point to an affection of any particular system or organ Such a scheme is necessarily v ry far from complete and may require to be supplemented in individual cases One is not able in such a work as this to explain *why* such and such questions should be put in affections of this or that organ or system The reasons for the questions the student will find out for himself Our present object is to help him in the interrogation of his cases so that he may not miss any important facts

1 Alimentary system and abdomen

(*a*) Symptoms point to an affection of the upper alimentary tract Inquire regarding—

Pain—What is its severity and exact site? Is it localized or diffuse? Does it radiate in any particular direction? For how long has he been subject to it? Has he had intervals of complete freedom? If so for how long? What is its relation to meals (if any)? Does it wake him at night? What things aggravate it? What affords relief e g food, alkaline powders vomiting?

Appetite—Is it excessive or diminished? If diminished, is his appet really bad, or is he afraid to eat on account of pain?

Meals—Arrangement of these the nature of the food. Is the diet adequate in amount and in essential constituents?

Sensations referred to stomach—Their nature and where exactly they are felt Their relation to the taking of food are they produced or relieved by it? How long after food do they come on? Are they

specially influenced by different kinds of food? Distinguish especially between pain and mere sense of discomfort or fullness

Vomiting—Frequency by day or by night in the morning or in the evening Its relation to food is it only after food or does it occur at other times? Its relation to pain does it relieve pain or not?

General characters of vomited matter—Its amount and colour Is there ever "coffee grounds vomiting is it ever sour and frothy? Does it contain residues of food taken days before?

Flatulence—Relation to particular articles of food Does the flatus tend to escape downwards or upwards? Does either form relieve the symptoms?

Water brash—Does he ever experience excessive secretion of saliva into the mouth with regurgitation of mouthfuls of clear tasteless fluid?

Heartburn—Does he suffer from a burning sensation behind the lower end of the sternum?

(*b*) Symptoms point to an affection of the *intestines* Inquire regarding—

Diarrhœa—Number and time of occurrence of motions during the day their relation to meals or to special articles of food Colour of the motions are they formed unformed porridge like frothy or frankly watery? Has he ever passed any blood or slime? Is there any straining (tenesmus) during defæcation? Is there abdominal pain or pain at the anus during defæcation? Does the patient use purgatives or does he take anything else e g beer likely to produce loose motions?

Constipation—What is his usual habit? Are the bowels opened regularly and if so how often? Has there been recent change in bowel habit? How long since the last motion? Has he ever noticed any grooving or flattening of the motions? Does the constipation alternate with diarrhœa? If so can this be explained by the taking of purgatives? Has he any griping pain? Has he had any vomiting?

Pain—Site radiation and character? Persistent or intermittent? Where is it felt worst? Is it relieved by defæcation or by the passage of flatus?

(*c*) Symptoms point to an affection of the *liver* or *gall bladder*—e g patient is jaundiced or has pain in the region of the liver Inquire regarding—

Pain—Its site Has he ever had any attacks of very severe pain coming on suddenly and lasting for a few hours? If so did the pain radiate and in what direction? Was there vomiting with it? Was he yellow after it subsided? Has he ever had pain in the tip of the shoulder?

Has he noticed any change in the colour of the urine or fæces?

Inquire also regarding his digestion on the lines of the interrogation already laid down for affections of the stomach

2. The symptoms point to an affection of the circulatory system Inquire regarding—

A history of rheumatic fever chorea scarlet fever or diphtheria (If a child ask also about sore throats and "growing pains")

The following subjective sensations —

Dyspnœa —When does it come on? Is it present at rest or only on exertion? What degree of exertion is necessary to produce it? Does he have attacks of breathlessness at night? Has he to sit up in bed, or can he sleep lying down? *Præcordial pain* or distress its exact site and character does it radiate or not? If so in what direction? What precipitates it and what if anything relieves it? *Palpitation* what brings it on and how long does it last? Does the heart give an occasional thump now and then?

Ask for signs of general venous congestion—e g do the feet ever swell? Has he any cough? What is the state of the digestion?

3 The symptoms and appearances point to an affection of the blood Inquire regarding—

Family history of bleeders Has he had any loss of blood? Are the stools ever black? Has he bleeding piles? (If a woman—is *menstruation excessive or diminished?*) *What kind of a diet does he* eat? What drugs has he been taking and to what chemical substances is he exposed in his work or home?

Such subjective sensations as breathlessness on exertion headache giddiness palpitation.

Do the feet ever swell?

4 The symptoms point to an affection of the respiratory organs Inquire regarding—

Family history of phthisis The patient's occupation does it expose him to the inhalation of irritating fumes or particles? How is his appetite? Does he sweat at night? Is he getting thinner? How much does he smoke?

Cough —Its character and frequency when is it worst? Does it cause pain or not?

Expectoration —Its amount and general characters ever blood in it? If so how much?

Pain in chest —Where is it seated? Is it aggravated by taking a breath? Is it constant or not?

Dyspnœa —When is it felt? If spasmodic ask him to describe an attack

5 The symptoms point to an affection of the kidneys—e g œdema—or urinary passages—e g pain in micturition Inquire regarding—

History of scarlet fever tonsillitis or previous renal disease
Has he any pain in the lumbar region or any attacks of acute pain shooting down into the groin or testicles?
The following remote symptoms —
Headache vomiting drowsiness paralysis or fits dimness of vision dyspnœa
Does the face ever look puffy in the morning? Are the ankles swollen? What is the state of the bowels?

Inquire regarding micturition as follows —

Urine —Is it altered in amount? Has he to rise in the night to pass it?
Is it altered in colour? Is it clear or turbid when passed? Ever any blood in it? If so at what period of micturition is it present?
Is there any increased frequency of micturition? Is the increase by day or by night? Is there an increase in volume passed?
Is there any pain during micturition? Is it before during or after the act? What is its character and where is it felt? Is it aggravated by movement?

6 In skin diseases

Inquire carefully into the patient s personal habits as regards diet clothing and washing What is his occupation? Does he handle chemical substances or other irritants? Ask if he has been taking any drugs recently It may be nec ssary to inquire carefully regarding syphilis Does the eruption itch? If so when is the itching worst? Did the eruption appear all at once or in crops? Does he suffer from asthma hay fever or any other allergic conditions?

7 The symptoms point to an affection of the nervous system Inquire regarding—

A *family* history of mental disease paralysis or fits
The nature of the patient s work is he exposed to any poisons—e g lead, mercury manganese carbon bisulphide or other volatile substances? Syphilis and alcohol should be inquired about with special care Has he been exposed to tropical infestations?
In cerebral cases it is important to inquire regarding discharge from the ear

Should he complain of *fits* the following questions should be asked —

Age at first fit? Any assigned cau e? Describe the first fit When did the second occur? What has been shortest and longest interval between the fits? Are they more or less frequent now? Do they occur in sleep or not? Has he any premonition or aura? What is its character? How long before the loss of consciousness does it occur? Is the onset sudden or gradual? Are convulsions present? Are they general or local? Where do they begin and end? Does he fall? Has he ever hurt himself? Does he bite his tongue micturate or defæcate during the fit? Are there any after symptoms such as sleep headache automatism or paralysis? Is there any subsequent mental disturbance? Because these patients are seldom clear as to the exact nature of their fits it is essential to interview separately a reliable person who has seen the patient in a fit The word fit is undesirable at a first questioning and attack is preferable

If he complains of *paralysis* inquire regarding—

Symptoms of heart disease hypertension or diabetes (*see* Circulatory and Urinary Systems) Had he any premonitory symptoms before the onset? How did the paralysis come on? Suddenly or gradually? Has he any headache or vomiting? Where is the headache situated? Has he any giddiness or difficulty in walking? (The method of eliciting other subjective symptoms of nervous disease is considered along with the investigation of the cranial nerves in Chap IX p 278)

8 The following special scheme for cases presenting **mental symptoms** will often be found useful —

The mental condition of the patient is assessed by (1) what we observe of his behaviour (2) by the way he responds to our questions (3) by the information which is given us by his relatives and friends Points about the behaviour of the patient are alone mentioned here Our picture of him rests not only on how he behaves but on how he tells his own story

Is he reasonably clean and neat or unkempt of hair nails and un shaven?

Is he reacting in the expected way to his complaints or is he over talkative or almost mute? Does he display unwarranted hilarity fussiness or resentment? Is he placid or excited? Are both his conversation and his movement slower or quicker than one would expect? Does he comprehend where he is or is his consciousness clouded—slightly seriously (state of stupor) or is he unconscious (coma)? Is he incontinent of fæces or urine?

Is he idle active co-operative or not? Can his attention be held

or does it wander? Does he answer questions directly or slowly or make unexpected replies?

Does it appear that he is intelligent or dull witted? Is the story which he tells to the point or discursive and vague and is this due to some temporary clouding or permanently poor mentality?

Any other information suggesting a disturbed mental state should be included in the clinical note The detailed way in which a fuller psychiatric examination is to be conducted will be found on p 270

9 The symptoms point to an affection of the bones or joints

Inquire specially in the family history for tuberculous disease and in the personal history for tuberculous disease previous manifestations of gout or rheumatism for syphilis or gonorrhœa and for any remote or recent injury (and in a woman for leucorrhœa)

If there is pain referred to a bone ask whether it is worse during the day or during the night If the pain is in a joint ask whether it is present constantly or only when the joint is moved Does the pain shift from one joint to another?

10 The following notes on taking of an occupational history will be found helpful

In clinical practice the occupational history is often valuable and there are few surer and quicker means of gaining a patient s confidence than the display of an intelligent knowledge of his job It is a wise rule to take the occupational history from the time the patient left school Record the dates and items of all subsequent jobs He may be exposed to a noxious substance responsible for his ill health in his present occupation but this should not be assumed A man describing himself as an ice-cream vendor may have cancer of the skin of the hand due to work in the pitch beds of a gas works 20 years before Ask the patient the name of his trade the processes employed the tools used and the substances handled The name of an occupation may be misleading for different names are used for the same process in different parts of Great Britain

It often happens that workmen and foremen refer to chemical substances by their popular names and not by their chemical names Examples of such names are lunar caustic for silver nitrate chrome yellow for lead chromate wood spirit for methyl alcohol and oil of mirbane for nitrobenzene The man may be ignorant of the nature of a substance he uses and know it only by a trade name In such cases it is best to communicate with his works manager and ask what is the nature of the substance in question

Question him as to the general conditions at his place of work If necessary ask him to sketch on paper a plan of his workshop and of

the apparatus he uses Is the job dusty and if so what tools make the dust? Are there fumes or vapours and if so what are the chemical substances involved? Most of the toxic substances encountered in the dangerous trades enter the body by inhalation Ask whether a hood is installed over his bench and whether it is connected to a suction system. Ask about the provision of protective clothing at his place of work Does he wear a special suit gloves or goggles and why? Finally ask whether any similar illness has befallen a fellow workman

Whenever serious doubts and difficulties arise it is advisable to visit the factory in order to ascertain the conditions of work on the spot In difficult cases the practitioner should enlist the help and advice of the Factory Department through H M Senior Medical Inspector of Factories Ministry of Labour and National Service 8 St James s Square London S W 1

Other aspects of the history are no less important A particular illness may render a man temporarily or permanently unfit to do his work The doctor should know that conditions peculiar to certain trades may cause disease which predisposes to infection Thus silicosis leads to an excessive mortality from pulmonary tuberculosis and also from pneumonia Diseases other than infections may be involved For example a heavy mortality from cirrhosis of the liver as well as from tuberculosis exists among publicans barmen brewers draymen and others who have ready access to alcohol

The doctor should have regard for his patient s work even when he is suffering from a disease which is non occupational one must know whether a man does a job which makes him a danger to others A dairyman with open tuberculosis can contaminate milk with tubercle bacilli by coughing into it and those who handle food can initiate out breaks of typhoid fever dysentery and cysticercosis by acting as carriers

11 If the patient is a young child the following special questions should be put to the mother or other responsible person —

How many other children are there? Any dead and of what? Where does patient come in the family? Have there been any miscarriages? If so when? Health of father s and mother s family? Mother s health during pregnancy

Was this a full time child? Was the labour normal? Was the child breast fed if so how long? If not how was it fed? When did mixed feeding begin? What food does it get now? Had it any rash after birth or any snuffles? When did it begin to get its teeth to walk and to talk?

What is the usual state of the digestion and bowels?

Inquire regarding previous illnesses Infectious diseases and ages at which they occurred (measles whooping-cough chicken pox scarlet

fever etc) Fits (number and dates) attacks of diarrhœa vomiting sore throat or bronchitis Has there been any running from the ears? Have there been severe persistent limb or joint pains? If the child has a cough inquire whether it has ever whooped when the attacks are worst and whether the cough is ever followed by vomiting

The interrogation of the patient being completed proceed to—

II THE PHYSICAL EXAMINATION

Investigate first the patient s general state This includes the general condition of his nutrition the presence of any obviously morbid appearances and other points considered in detail in Chap II After that proceed to an examination geographically from above downwards afterwards writing down the findings under the various systems The results yielded by inspection palpation percussion and auscultation should always be stated in that order

One more point regarding case taking remains to be emphasized and that is to note negative as well as positive facts It is often quite as essential for example to state that such a symptom as dyspnœa is absent as to record the fact of its presence This is a point which may not be fully appreciated by the beginner

It need hardly be said that the examination should be carried out as gently as possible all unnecessary exposure exhaustion or chilling of the patient being carefully avoided If the patient is suffering from severe or acute disease it may be advisable to postpone all physical examination other than that which is absolutely necessary for the diagnosis of his condition or for guidance in treatment Remember that when a patient is much exhausted or suffering from serious disease of the lungs or heart very dangerous and even fatal results may ensue if he is thoughtlessly made to sit up in bed in order to have his chest examined

CASE TAKING SCHEME

I INTERROGATION

Name Age Occupation Married or single Address Date of coming under observation

Presenting complaint

Duration

Family history

Inquire regarding parents brothers and sisters and patient s own children Note state of their health or the case of their death with age at which they died

Personal history

Social history —Environment nature of work and its surroundings *Hygienic conditions at home Mental attitude interests hobbies* fears and ambitions

Previous illnesses or accidents (if any) with their time of occurrence duration and results

Habits as to exercise food alcohol and tobacco

Present illness —Time and mode of its origin the order in which symptoms appeared and the chief symptoms which trouble patient now treatment (if any) already employed

II PHYSICAL EXAMINATION

General

While taking the history and in the course of the examination the following points should be observed —

General aspect of the patient appearance of illness
intelligence
co-operation
expression
position
build

Temperature and pulse rate
Skin colour cyanosis anæmia jaundice pigmentation
Skin eruptions
Œdema wasting obesity
Body hair
Deformities swellings

The main examination begins with the head and neck and progresses down the body

The Head and Neck

Skull shape percussion and auscultation
Hair
Eyes Exophthalmos ptosis œdema of lids
Conjunctivæ anæmia jaundice inflammation

Pupils size equality regularity reaction to light and accommodation
Eye movements strabismus nystagmus
Fundi
Acuity of vision and visual fields
Ears Inspection of drums and simple tests of hearing
Face Function of motor and sensory parts of fifth nerve
Function of seventh nerve
Mouth and pharynx (a tongue depressor and torch should be used)
Breath
Lips colour and eruptions
Tongue protrusion and appearance
Teeth and gums
Buccal mucous membrane colour and pigmentation
Pharynx movement of soft palate state of tonsils
Neck Lymphatic glands
Venous distension and palpation of carotid vessels
Thyroid
Movements and muscle power

Upper Limbs

General examination of arms and hands
Finger nails clubbing koilonychia
Pulse rate rhythm volume character thickness of arterial wall of radials and brachials
Axillæ lymphatic glands
Blood pressure
Nutrition power muscle tone tendon reflexes co-ordination and sensation
Joints

Thorax

1 *Anteriorly and laterally*
Note the type of chest and any asymmetry
Rate depth and character of respiration
Pulsations
Dilated vessels
Define the position of the trachea by palpation
Look for the apex beat
Palpate apex beat
Palpate over precordium for thrills and over any visible pulsations
Define the area of cardiac dullness by percussion
Auscultate the heart sounds
Palpate respiratory movements
Estimate tactile vocal fremitus

Percuss the lungs
Auscultate the breath sounds
Estimate vocal resonance

2 *Posteriorly* (patient sitting)
Inspect and palpate chest expansion—apices and bases
Estimate vocal fremitus
Percussion of chest
Auscultation breath sounds vocal resonance
Note and palpate deformities of spine spinal movements and the presence or absence of tenderness œdema of lumbar pads
Palpate from behind cervical glands thyroid

Abdomen

Inspection size shape distension symmetry movement of abdominal wall dilated vessels scars umbilicus visible peristalsis pulsations pubic hair hernial orifices
Palpation tenderness rigidity hyperæsthesia splashing increased resistance to palpation masses fluid thrill liver spleen kidneys abnormal masses
Percussion when necessary
Auscultation when necessary
Hernial orifices patency impulse on coughing
Inguinal glands
Genitalia penis scrotum spermatic cord and testicles
Abdominal reflexes
Rectal examination
Vaginal examination if indicated

Lower Limbs

General examination of legs and feet
Œdema—pitting of skin over ankles and thighs
Nutrition power muscle tone tendon reflexes co-ordination and sensation
Joints Pulsation in dorsalis pedis and posterior tibial arteries

Examination of Excreta

1 Urine
2 Sputum
3 Stools } naked eye description including amount
4 Vomitus

In addition to the above scheme a full neurological and/or psychological examination may be necessary Neurological examination is described in Chap IX and a brief outline of psychological examination is included in this chapter

Writing out the physical examination

The date must be given. *Whatever the order in which the examination was performed it should be described systematically. Relevant negative* findings are as important as positive ones.

General.—Begin with a description in one or two sentences of the general appearance and mentality.

Record the temperature.
Record the state of —
Mucous membranes
Skin
Mouth
Nails
Hair
Thyroid gland
Lymphatic glands
Bones and joints

Note the presence or absence of anæmia, cyanosis, jaundice, œdema, clubbing of the fingers, and the condition of tongue, fauces, tonsils and teeth.

Cardiovascular System
Pulse
Condition of other arteries
Venous engorgement
Pulsations and their character
Heart.—*position* of apex beat and its character; presence or absence of thrills; character *and intensity of* heart sounds; murmurs and their propagation
Blood pressure

Respiratory System
Rate and character of *respiration*
Position of trachea
Shape of chest: symmetry
Results of inspection, *palpation*, percussion and auscultation, noting areas affected if any abnormality is found
Sputum: quantity and character

Abdomen
Inspection: *shape*, *distension* (general or local), movements with respirations, venous distension, *umbilicus*, *pulsation*, visible peristalsis
Palpation and percussion: tenderness, rigidity, hyperæsthesia



Cretinism



Mongolism



Dehydration



Tetanus

Plate 1 —FACIES

Writing out the physical examination

The date must be given Whatever the order in which the examination was performed it should be described systematically Relevant negative findings are as important as positive ones

General —Begin with a description in one or two sentences of the general appearance and mentality

Record the temperature
Record the state of —
Mucous membranes
Skin
Mouth
Nails
Hair
Thyroid gland
Lymphatic glands
Bones and joints

Note the presence or absence of anæmia cyanosis jaundice œdema clubbing of the fingers and the condition of tongue fauces tonsils and teeth

Cardiovascular System
Pulse
Condition of other arteries
Venous engorgement
Pulsations and their character
Heart —position of apex beat and its character presence or absence of thrills character and intensity of heart sounds murmurs and their propagation
Blood pressure

Respiratory System
Rate and character of respiration
Position of trachea
Shape of chest symmetry
Results of inspection palpation percussion and auscultation noting areas affected, if any abnormality is found
Sputum quantity and character

Abdomen
Inspection shape distension (general or local) movements with respirations venous distension umbilicus pulsation visible peristalsis
Palpation and percussion tenderness rigidity hyperæsthesia

liver spleen kidneys any other abdominal masses ascites hernial orifices
Auscultation peristaltic sounds
Spine
In men genitalia rectal examination
In women vaginal and rectal examination as indicated

Nervous System
General Mental state intellectual functions emotional state speech and articulation gait
Cranial nerves In any neurological case a full description of the examination of the cranial nerves taken in their order should be given
Upper limbs
Trunk
Lower limbs
Physical signs should be described in the following order — Wasting and fasciculation power tone co ordination reflexes sensation

Urine
Colour reaction specific gravity sugar albumin blood deposit (microscopic examination)

Stools Sputum and Vomit should be described

Diagnosis

(Prognosis)

Notes of Treatment and Progress

(Daily notes in acute cases in others make a note of progress every three days)
State on dismissal

If patient died add notes of post mortem (if held)

CHAPTER II

GENERAL CONDITION AND APPEARANCES

It should hardly be necessary to say that a proper physical examination demands the absence of noise and an adequately warmed room Good daylight is essential for the perception of colour changes *such as those present in anæmia and cyanosis while slight degrees* of jaundice cannot be seen at all in artificial light For a complete physical examination the patient should be stripped completely and covered with a blanket or dressing gown though only the part or parts actually being examined need be uncovered at any one time Even so if the examination is attempted in a cold room the patient usually shivers This is distressing to him and makes most forms of examination useless In particular it is impossible to auscultate the chest as strange noises are produced in the stethoscope by shivering muscle Ideally another woman should be present when a male doctor is making any examination of a female patient and this is essential in the case of rectal and vaginal examinations both to reassure the patient and to protect the doctor from subsequent accusations of impropriety

Physical examination proceeds by the time honoured methods of inspection palpation percussion and auscultation applied with appropriate modifications to the different systems and parts of the body It may be said however that the experienced doctor begins his examination as soon as the patient enters the room just as he continues taking a history till the patient leaves it Examination of systems may provide information about the state of organs and functions but it is also important to try and view the patient as a whole

Thus from the moment of meeting the patient and while taking the history the experienced doctor will be making observations The patient s gait as he walks into the room his posture when sitting or standing his dress speech demeanour manner of answering questions level of intelligence and emotional state may all yield valuable information What kind of a person is this? What is his background? What is he thinking? What is he

feeling? Is he in general a happy person or an unhappy one? He is probably anxious Is his anxiety proportionate to the sign of disease present and reasonable or disproportionate and un reasonable? What is the real reason for his coming to see a doctor at this particular time? Doctors vary in their ability to answer questions of this kind and the skill to do so can in any case only come with actual practice Students should realize that an important function of their periods of clerking in wards and out patient departments is to give them an early opportunity to acquire something of this ability which cannot be gained from any textbook or lecture

One of the first things to observe about a patient in bed is his **attitude** In health a person lies in any manner in which he feels comfortable He changes his position without much difficulty from time to time and has no hesitation in altering his attitude if he slips from his pillows or feels otherwise uncomfortable But the tress of disease will often confine his activity in narrow bounds When fever has run high or when some other cause has reduced the patient to extreme weakness and dulled his consciousness he no longer makes any effort to secure a position of comfort but *passively slips downwards* from his pillows and lies listless flaccid and silent even when the resulting attitude is such as to render the act of breathing unnecessarily exhausting

Patients with heart failure and congestion of the lungs are often unable to lie in a horizontal position and must sit more or less upright in a chair or propped up with pillows This difficulty in breathing when lying flat is referred to as orthopnœa

In abdominal disease the aspect of the patient is often character istic When peritonitis is present, he lies on his back still and quiet breathing very shallowly to allay the pain that movement produces Sometimes one or both legs are drawn up depending upon the extent of the inflammation

In colic there is often great restlessness which contrasts vividly with the fixed attitude of peritonitis in renal colic the patient rolls about and tries one position after another in futile search for a posture free from pain

Patients who are attacked by acute rheumatism have a peculiar aspect of helplessness the limbs lying motionless the joints being swollen stiff and painful

Various diseases of the nervous system produce characteristic

attitudes peculiarly important is that of meningitis where in the severest cases the neck is bent backwards so that the head seems to bore into the pillow

When possible the physician should not only study his patient in bed but should also see him up and walking Many very characteristic attitudes which are of the greatest value in forming a diagnosis can only be observed when the patient is in the erect posture Thus the forward stoop and festinant gait of paralysis agitans are as characteristic as the mask like facies tremor and pill rolling movements

When the patient is standing observe (1) the pose of the head (2) the set of the shoulders (3) the inclination at which the trunk is carried on the pelvis—thrown back in hypertrophic muscular dystrophy in pregnancy and in massive abdominal tumour—often bent forward when abdominal pain is present (4) the position of the arms and hands (5) the outline of the lower limbs

When the patient walks any peculiarity in his gait must be observed The more important types of gait are described in Chap XI but it should be remembered not only that alterations may be due to diseases of the muscular and nervous systems but that local conditions in toes heels or in any of the joints of the lower limbs may produce characteristic effects

Notice should be taken of the dress Apart from insanity where the patient s clothing is frequently dishevelled or grotesque one may discover indications of a local or general change in his bulk or his boots may wear unevenly in consequence of some abnormality of gait.

The general development and nutrition of the patient should be compared with the examiner s opinion of the normal for the patient s age sex and physical type In medical examinations for insurance purposes it is usual to record the chest measurement during inspiration and expiration at the level of the nipples (in male subjects) and also the waist measurement If the latter exceeds the former either the subject is unduly fat or the abdomen is en larged by disease usually by the presence of fluid (ascites) Wide variations in build are compatible with good health The following tables show ideal weights for different heights and builds

In assessing nutrition one notes whether the patient is too stout is well nourished or is emaciated In health there is a fair quantity

IDEAL WEIGHTS FOR MEN (AGES 25 AND OVER)

Height (with shoes)	Weight in Pounds (as ordinarily dressed) Small Frame	Medium Frame	Large Frame
ft. in.			
5 2	116-125	124-133	131-141
5 3	119-128	127-136	133-144
5 4	122-136	130-140	137-149
5 5	126-136	134-144	141-153
5 6	129-139	137-147	145-157
5 7	133-143	141-151	149-162
5 8	136-147	145-156	153-166
5 9	140-151	149-160	157-170
5 10	144-155	153-164	161-175
5 11	148-159	157-168	165-180
6 0	152-164	161-173	169-185
6 1	157-169	166-178	174-190
6 2	163-175	171-184	179-196
6 3	168-180	176-189	184-202

IDEAL WEIGHTS FOR WOMEN (AGES 25 AND OVER)

Height (with shoes)	Weight in Pounds (as ordinarily dressed) Small Frame	Medium Frame	Large Frame
ft. in.			
4 11	104-111	110-118	117-127
5 0	105-113	112-120	119-129
5 1	107-115	114-122	121-131
5 2	110-118	117-125	124-135
5 3	113-121	120-128	127-135
5 4	116-125	124-132	131-142
5 5	119-128	127-135	133-145
5 6	123-132	130-140	138-150
5 7	126-136	134-144	142-154
5 8	129-139	137-147	145-158
5 9	133-143	141-151	149-158
5 10	136-147	145-155	152-166
5 11	139-150	148-158	155-169

(Metropolitan Life Insurance Company Statistical Bureau 1943.)

It will be noticed that modern tables of this kind make no allowance for so-called middle aged spread

of subcutaneous fat the muscles are of moderate size and firm texture whilst those which have been called into special exercise in the ordinary occupation of the individual under examination may be unusually well developed The skin is elastic and neither very moist nor very dry When nutrition is disordered either the muscles become flabby and the subcutaneous fat is increased so as eventually to become burdensome to its possessor or emaciation sets in Emaciation occurs in the late stages of many diseases especially malignant growths pulmonary tuberculosis thyrotoxicosis and diabetes mellitus

To the trained observer the expression of the patient yields information of the very highest importance and amongst the factors which determine expression the eye holds the foremost place Some patients cannot look their doctor in the face and this tendency to avoid catching his eye is important possibly indicating that the information they are about to give lacks truthfulness It may however simply be due to self consciousness Sometimes the eyes are restless following every movement of the attendant as often occurs in phthisis at other times they stare vacantly regardless of all that is passing around them—a condition well seen when the consciousness is growing dull In exophthalmos the eyes are prominent often showing a ring of sclerotic above the cornea or the prominence may be due to a high degree of myopia In wasting disease or in profound collapse such as is found in cholera the sunken eyes and half-closed eyelids cannot fail to command attention

The lower eyelids are puffy and œdematous especially in the morning when the patient is suffering from acute nephritis and a like appearance is often to be noted in patients who are suffering from severe paroxysms of cough It is characteristically present in children affected with whooping-cough

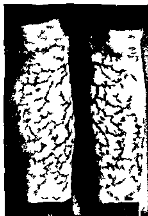
The nose has a sunken bridge in congenital syphilis the tip is red in some cases of mitral stenosis in some females with chronic indigestion and occasionally in purely local conditions Undue mobility of the alæ nasi may be due to neurosis or may indicate obstruction to inspiration and is in this respect very important to look for in infants Young persons who suffer from adenoids and to a lesser extent those afflicted with enlarged tonsils or chronic bronchitis have pinched noses and open fishy mouths The mouth is kept open to reduce resistance to the entry of air whilst the



I u I rm



Ultrav l t Irri l at n

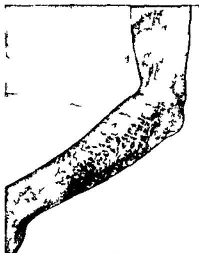


L vedo Ret cula is



A en I gmentation

Plate 3 —PIGMENTATION OF SKIN



I ll arm



All n D c n l n r al contr l

pinching of the nose is due to falling in of the alæ nasi where they lose the support of the nasal bones which do not develop properly under these circumstances

The lips are pale in anæmia livid and blue in congestive heart failure The vesicles of herpes febrilis on the lip are very often associated with inflammation of the respiratory tract and their presence should lead to a close search for pneumonia

The ears are often ill developed in idiots and sometimes hæma tomata develop in the insane Of greater frequency is the occur rence of tophi in persons of gouty habit

The cheeks give valuable information regarding the patient s health In anæmia and aortic regurgitation they are pale in some cases of mitral stenosis there is a bright circumscribed flush over the malar bones in many persons who lead an open air life they are ruddy and high-coloured in congestive heart failure they are also high-coloured but the colour is of a bluish tint which cannot be mistaken for the rubicund cheeks of weather beaten people

The form of the cranium may also indicate some points of importance to which reference is made in Chap XI

In addition to the appearance of individual features the general expression of the patient must be noted Is it animated apathetic or has it the absolute vacancy of unconsciousness? Are there wrinkles on the face or is it smooth or is one side smooth and the other wrinkled as one sees it in unilateral paralysis of the seventh nerve? Is the mouth drawn over to one side and is there any other lack of symmetry between the two halves? The expres sion may be characteristic of pain or may show a placidity gain saying assertions of severe agony An anxious aspect often presages severe illness at a time prior to the appearance of any other signs and symptoms Twitching of the face sometimes results from a nervous habit at other times it is a symptom of definite disease of which chorea affords a good example (Plates 1 2)

When pain is present facial features are often differently affected according to its situation Pain in the head whether simple head ache or of organic origin causes the sufferer to frown pain in the chest and abdomen tends rather to affect the expression of the lower part of the face These signs are of peculiar importance in the case of children who cannot describe their sufferings

The physiognomy of insanity is often characteristic but descriptions of it must be obtained from special textbooks In serious illness the nose is often pinched the eyes sunken and lustreless and the chin and malar bones sharp and prominent

The **state of the skin** where it is exposed must be carefully investigated In the face we notice especially the **complexion** This is dependent on two factors—the colour and the transparency of the skin The most important abnormalities are pallor yellowness pigmentation and cyanosis **Pallor** occurs in anæmic states and also in fainting or severe nausea Anæmia however is to be judged by the colour of the blood rather than that of the patient and the colour of the skin may be most misleading That of the mucous membranes of the mouth and conjunctivæ gives a better indication **Yellowness** may be due to hæmolytic jaundice when the tint is pale lemon yellow or to obstructive jaundice when it may be of a dark yellow or orange colour In obstructive jaundice there may be excoriations from the scratching that results from the intense itching which the bile salts evoke **Pigmentation** is found in Addison s disease where it affects both the skin and the buccal mucosa Other forms of pigmentation are described in Chap VIII The **cyanosis** of embarrassed breathing and of heart failure does not demand further notice here

It is also important to search for **cutaneous eruptions** some of which—measles and syphilitic rashes for example—frequently appear first about the roots of the hair whilst others have equally distinctive situations Ulcers and scars should be sought The colour and nutrition of the hair and the dryness or moisture of the skin must be noted and if perspiration is present its amount and situation The perspiring hands of a rheumatic subject are very characteristic

When an excess of fluid is present in the subcutaneous tissue the condition is known as **œdema** Thus in acute nephritis an early symptom is œdema of the face especially below the eyes which comes and goes being most noticeable when the patient rises in the morning In dependant œdema however which is typically present in congestive heart failure and in conditions associated with a low plasma protein level the swelling first appears at the ankles and over the dorsum of the foot and only gradually mounts to the legs thighs and trunk In local venous obstruction the œdema is confined to the parts from which the return of blood is



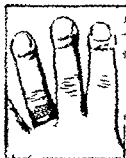
Rheumatoid Arthritis



Heberden's Nodes



Koilonychia



Clubbing

Plate 5 —HAND



Collateral V nous Anastomos



Topl f Co t



R k t Rosary

Plate 6

impeded In this way one finds œdema of an arm when cancerous axillary glands constrict the axillary vein or œdema of a leg in thrombosis of the popliteal or femoral vein Œdema of the whole upper part of the body may result from intrathoracic tumours or more rarely from compression of the superior vena cava by an aneurysm Œdema may be recognized by the pallid and glossy appearance of the skin over the swollen part by its doughy feel and by the fact that it pits on finger pressure

Localized œdema may be due to local changes in capillary per meability as in angio neurotic œdema and giant urticaria

Subcutaneous emphysema is uncommon but if present can be readily recognized by the crackling sensation which is detected on pinching the part affected

The **hands** of the patient merit careful observation (Plate 4) Notice the strength of grip as he shakes hands this often indicates improvement or deterioration with considerable accuracy Their general shape should be noted along with the state of the joints the character of the nails and the presence or absence of finger clubbing In osteo arthritis the finger joints are often implicated and nodules known as **Heberden s nodes** are formed at the terminal joints In nerve disease the skin of the hand may undergo trophic changes becoming thin and glossy or the vessels may be in fluenced by vaso motor disorders leading to redness or to a pallid and dead looking state of the fingers Characteristic movements or attitudes of the hand may also be seen in athetosis tetany and lead palsy Tremor of the hands may occasionally be congenital In other cases it is due to nervousness senility Parkinsonism thyrotoxicosis alcoholism disseminated sclerosis uræmia hepatic failure *or mercurial poisoning The methods of studying this symptom* are detailed at p 322 In ulnar paralysis the hand becomes deformed by over-extension of the first phalanges combined with excessive flexion of the rest so that a claw like attitude is produced This is known as the **main en griffe** When the muscles of the thenar and hypothenar eminences have undergone atrophy the hand becomes flattened and thus somewhat simian In acromegaly the hands are massive the fingers being spatulate with square tips the knuckles enlarged and the skin thickened **In clubbing of the fingers** the tissues at the base of the nail are thickened and the angle between the base of the nail and the adjacent skin of the finger is obliterated The nail itself loses its longitudinal ridges and becomes

convex from above down as well as from side to side In extreme cases the terminal segment of the finger is bulbous like the end of a drumstick The condition may be congenital or acquired Gross degrees of clubbing are found in association with severe chronic cyanosis as in congenital heart disease and in association with chronic suppuration within the chest as in bronchiectasis and empyema Minor degrees may be found in pulmonary tuberculosis and in chronic abdominal conditions such as steatorrhœa and ulcerative colitis In **hypertrophic pulmonary osteo arthropathy** there is besides clubbing of the fingers thickening of the periosteum of radius ulna tibia and fibula This gives rise to swelling above the wrist and ankle *In rare cases these joints themselves are swollen* A transverse furrow in the nail is the record of some former interference with its nutrition and in the absence of a local cause may indicate some severe illness in the recent past **Koilonychia** occurs in idiopathic hypochromic anæmia The nails are soft thin and brittle The normal convexity is entirely lost and replaced by hollowing so great that in some cases eight drops of water can be run into the concavity of the nail without overflowing In infants the movements or position of the hands and fingers will often direct an acute observer to the seat of disease

The neck should always be inspected and special note taken of any of the conditions described in the paragraphs that follow

1 **The state of the lymphatic glands** In secondary syphilis the glands under the upper part of the trapezius are often palpable In infected conditions of the tonsils the glands at the angles of the jaw are enlarged and those below the jaw in cases of malignant disease in the mouth Glands draining an inflammatory focus are usually tender Enlarged tuberculous glands may occur in groups or in long chains beside the sterno mastoid and scars will mark the points of past suppuration In Hodgkin s Disease and other reticuloses the glands are enlarged and discrete In lymphatic leukæmia there may be great enlargement of the glands on both sides If enlarged glands are found either in the neck or elsewhere it is important to observe whether they are firm and distinct fused together or whether fluctuation can be elicited

2 **The thyroid gland** Inspect the neck for any general or local enlargement of the gland and observe its movement with the larynx as the patient swallows Then stand behind the patient and palpate the gland with one hand on each side of the neck Determine if any

swelling exists and if so whether it is uniform or nodular hard or soft Sometimes such enlargements exercise considerable pressure on the trachea and occasionally extend into the thorax behind the sternum at other times particularly if the disease is malignant the recurrent laryngeal nerves may become implicated In cases where there is difficulty in determining whether a tumour is connected with the thyroid it is helpful to remember that the gland and any tumour connected with it moves up and down during deglutition

3 **Pulsations in the vessels of the neck** must be recorded Any arterial pulsation is both seen and felt as a distinct thrust whereas venous pulsation is seen but is not felt as a thrust if it is felt at all In aortic incompetence the carotid arteries are seen to pulsate forcibly Women patients with hypertension sometimes show kinking of the right common carotid artery which simulates aneurysm The jugular veins may be distended in congestive heart failure In superior mediastinal obstruction due to retro-sternal goitre or malignant neoplasm in the mediastinum distended veins may be seen all over the neck cyanosis and œdema may accompany *this sign*

4 **Boils and carbuncles** are very frequently situated on the back of the neck As they are not infrequently present in cases of diabetes the urine should be tested for sugar

The character of a patient s respiration is often of great service in diagno is and prognosis When the respiratory passages are obstructed the normal quiet respiratory sound is replaced by more or less noisy breathing When the obstruction occurs in the nose the breathing is sniffing or bubbling in character When the soft palate is relaxed and especially if paralysed a snoring stertorous sound is produced When the rima glottidis is obstructed from any cause such as spasm or paralysis of the vocal cords or œdema of the larynx stridor results If a polypus or other tumour lies between the cords there may either be stridor or simply noisy breathing The trachea may have its airway narrowed by pressure from the outside as in cases of tumour and especially of aneurysm when the breathing becomes growling or mucus may obstruct the lumen producing a rattling sound The death rattle which occurs when weakness and insensitiveness combine to prevent any effort at expectoration is a typical example of the condition Obstruction in the bronchi sometimes gives rise to audible wheezing and crackling sounds A division of dyspnœic conditions may be made according

as the difficulty in respiration is felt during the inspiratory or the expiratory period Most cases of obstruction of the large air passages are characterized by *inspiratory dyspnœa* whilst many of the pulmonary causes of dyspnœa produce *expiratory difficulty* Common examples of the latter are the prolonged expiration in bronchitis with emphysema and in asthmatic attacks The breathing may be characteristic of diseases quite distinct from those of the respiratory system Examples of this are the stertorous breathing of apoplexy the hissing expiration of uræmia and the air hunger of diabetic coma which affects both inspiration and expiration

If cough is present its character must be carefully noted It is important to observe whether the cough consists of independent explosive expirations or is paroxysmal The former occurs in early pulmonary tuberculosis in pharyngitis and in some forms of nervous irritation the latter is often found in severe bronchitis and is typical of pertussis In *bronchitis* the cough is at first short and dry but as the quantity of secretion increases it becomes more paroxysmal and continues until the mucus is expectorated When due to *early tuberculosis* the cough is frequent short and sharp It is described as *dry* because there is no rattling of mucus associated with it Later when the caseous masses are breaking down secretion is more copious and the cough becomes moist and paroxysmal In severe cases actual vomiting may be induced A *nervous cough* generally has the character of single short dry explosions repeated at intervals Local conditions in the *throat* may be the cause of most troublesome and persistent coughing and the observer should look for pharyngitis when the patient complains of constant hawking

In *pleurisy* and *pneumonia* (associated as it often is with pleurisy) the cough consists of solitary dry hacking expulsive efforts suppressed as much as possible to prevent unnecessary pain but repeated frequently In *laryngitis* and *croup* the cough may be simply noisy but more often is either husky or stridulous When the lumen of the trachea is encroached upon by a *mediastinal tumour* or an *aneurysm* there is generally a very resonant brassy cough aptly compared to a gander s cry When once heard this is almost sufficient to clinch the diagnosis without further examination

In *hysteria* the cough is often loud and barking and gives the impression of being produced to attract attention Such a cough

is sometimes associated with hysterical aphonia *Pertussis* when fully developed is distinguished by a most characteristic cough There is first a long-drawn almost stridulous inspiration then a series of short sharp expiratory coughs which follow each other with extreme rapidity The face turns dark and the veins grow prominent the child clings firmly to any support it can find so as to give full play to the accessory muscles of respiration and when at last the fit of coughing ends it is followed by a long drawn whooping inspiration The severity of the paroxysm induces vomiting and sometimes causes evacuation of the bladder and bowel

Hiccough which results from spasmodic contraction of the diaphragm is a common disorder It is usually due to trivial causes it also may occur in affections of the brain stem the stomach the peritoneum and in some general disorders the most common of which is uræmia In these conditions the hiccough is usually persistent

The voice as well as the cough should be studied The chief points to observe are its strength its clarity and whether aphonia exists The voice may be nasal either through habit or in consequence of obstruction in the upper airways

Temperature —When taking the temperature the following practical points must be attended to —

1 The thermometer must be accurate and of good quality

2 The thermometer must be kept in position long enough to allow the mercury to reach the body temperature It is well to exceed the period which the instrument professes to require The ordinary half minute thermometer should be left in position for one to two minutes

3 In adults the temperature is taken in the mouth or in the axilla in young children the thermometer should be placed in the fold of the groin and the thigh flexed on the abdomen or it may be inserted into the rectum The temperature of the mouth and rectum is generally at least half a degree higher than that of the groin or axilla When the temperature is taken in the mouth the patient must breathe through the nose and keep the lips firmly closed during the observation

4 Before inserting the thermometer make it an invariable rule to wash it in lotion or in cold water and see that the mercury is

well shaken down Wash it again before replacing it in its case In Great Britain the Fahrenheit scale is used on the Continent the Centigrade *

Normal	98 99 F	or 36 6 -37 2 C
Subnormal	Below 98 F	or below 36 6 C
Collapse	96 F	35 5 C
Febrile	Above 99 F	or above 37 2 C
Hyperpyrexia	107 F	41 6 C

In many conditions notably acute fevers there is a disturbance of heat regulation which may be looked on as the setting of the thermostatic mechanism controlling heat gain and loss at a higher level than normal While the temperature is rising to this new level heat is being conserved the skin vessels are constricted so that the body surface feels cold and the patient may even shiver violently This shivering is referred to as a **rigor** When the higher temperature is reached heat loss again becomes apparent the skin vessels dilate and the body surface feels warm This is the state of affairs present in sustained fever or **pyrexia**

There are three principal **types of fever**—the continued the remittent and the intermittent When fever does not fluctuate more than about a degree and a half (Fahrenheit) during the twenty four hours but at no time touches the normal it is described as **continued** When the daily fluctuations exceed two degrees it is known as **remittent** (Fig 1) and when fever is only present for

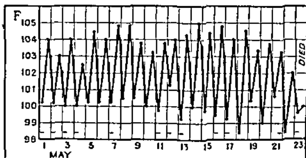


Fig 1 —Remittent fever (hectic) Case of pulmonary tuberculosis

* For a comparison of the two scales see Appendix 4 p 425

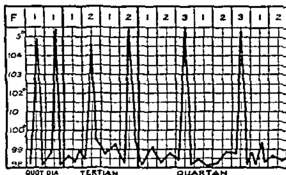


Fig 2 —Intermittent fevers

several hours during the day it is called intermittent In remittent fever the evening temperature is usually higher than the morning one but in some cases not infrequently in pulmonary tuberculosis this type is *inverted* and the remission occurs in the evening whilst there is a morning exacerbation When a paroxysm of intermittent fever occurs daily the type is quotidian when on alternate days tertian when two days intervene between consecutive attacks quartan

When the fever ends rapidly it is said to resolve by crisis (Fig 3) when gradually by lysis (Fig 4)

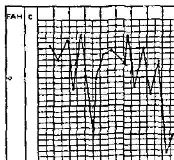


Fig 3 —Crisis Case of lobar pneumonia

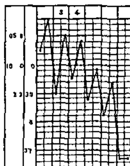


Fig 4 —Lysis Case of broncho-pneumonia

In the study of any case of fever the points which require to be observed are the height of the temperature and what its course has been what are the rate and character of the pulse whether the skin is moist or dry or exhibits any eruption and which of the viscera or secretions are characteristically affected The explanation of these points may be found in works on medicine but their true significance can only be learned at the bedside

CHAPTER III

THE ALIMENTARY SYSTEM AND ABDOMEN

I THE MOUTH THROAT AND ŒSOPHAGUS

THE MOUTH AND THROAT

The mouth —For the examination of the mouth the patient should be placed facing a good light If artificial light is used it should be thrown into the mouth by means of a reflector or a pocket torch

The lips —Note the colour of the lips They are blue in cyanosis pale in anæmia Note the presence of any crusts fissures or ulcers The lips should be everted to examine their inner surfaces Herpes febrilis of the lips is often seen in inflammatory conditions of the air passages and lungs especially in lobar pneumonia

The teeth —The temporary teeth are cut in the following order

First —The two lower central incisors sixth to eighth month
Second — The four upper incisors eighth to tenth month
Third —The lower lateral incisors and all the front mola s twelfth to fourteenth month
Fourth —The canines (upper first) eighteenth to twentieth month.
Fifth —Posterior molars at two to two and a half years

The permanent teeth appear as follows —

First molars at six years
Central incisors at seven years
Lateral incisors at eight years
Bicuspid (anterior) at nine years
Bicuspid (posterior) at ten years
Canines at eleven to twelve years
Second molars at twelve to thirteen years
Third molars at seventeen to twenty five years

The following table shows the numbers of the permanent and the temporary teeth —

		M	C	I	I	C	M	
TEMPORARY	Upper	2	1	2	2	1	2	20
	Lower	2	1	2	2	1	2	

		M	BI	C	I	I	C	BI	M	
PERMANENT	Upper	3	2	1	2	2	1	2	3	32
	Lower	3	2	1	2	2	1	2	3	

Observe the presence of any irregularity or defect or carious disease in the teeth The absence of a sufficient number of opposing and efficient molars may lead to improper mastication and so to digestive disorders Notice whether there is any exposure of the roots or whether they are surrounded with tartar Grinding of the teeth leads to bevelling of their edges this is especially found in nervous children The presence of Hutchinson s teeth affords (Plate 7) evidence of congenital syphilis In this condition the two central upper *permanent* incisors are rounded in section and slope inwards below they are broader nearer the gum than at the crown so as to be peg shaped and they present a semilunar notch at their biting edges They are usually discoloured as well In the same condition the first permanent molars tend to be dome shaped Enlargement of the lower jaw in acromegaly leads to alteration of the bite so that the lower teeth close outside the upper ones

The gums —Their colour should be noted In patients exposed to lead compounds a blue line can often be observed running along the edge of the gum especially opposite those teeth showing pyorrhœa This line must be distinguished from similar discoloration due to a black layer of tartar on the teeth If a wedge shaped slip of white paper is inserted between the gum and the tooth the stippled line of lead poisoning will be rendered more distinct whereas discoloration due to tartar on the teeth will disappear A similar but more diffuse line may be seen after a course of intramuscular bismuth preparations The gums may be swollen and spongy in scurvy (Plate 7) Hypertrophy of the gums may occur in pregnancy and in epileptics treated for long periods with sodium phenytoin (Epanutin) Hæmorrhages may be observed in the buccal mucous membrane in thrombopenic purpura and acute



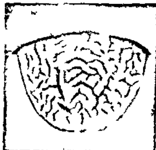
Hut hinson T th



Gun s n Scurvy



Lingual H n i trophy



C n nit l Fissur

Plate 7 MOUTH

leukæmia In *gingivitis* the edges of the gums are red and bleed easily In *pyorrhœa* the gums are retracted and frequently bleed easily Pus may be observed exuding from between the gums and the teeth or may be squeezed up by pressure on the gums

The tongue —Ask the patient to protrude it Slight deviation from the mid line is not uncommon and may be due to asymmetry of the jaws In hemiplegia, deviation towards the paralysed side may be found while in lesions of the hypoglossal nerve or its nucleus there may be fibrillation of the affected side Later this side may be wasted and deeply grooved—lingual hemiatrophy (Plate 7) Tremor of the tongue may be due to nervousness thyrotoxicosis delirium tremens or dementia paralytica

Next examine the surface of the tongue and note (1) its colour Is it pale red or discoloured? Pallor is seen in severe anæmia A fiery sore tongue usually indicates a nicotinic acid deficiency A slightly swollen magenta coloured tongue is found in riboflavin deficiency (2) Is it dry or moist? The state of the tongue is a valuable indication of the state of hydration of the body A dry and shrivelled tongue usually indicates a need for an increased administration of fluid A dry brown tongue may be found in the later stages of any severe illness but is found particularly in uræmia and acute intestinal obstruction (3) The presence or absence of fur Furring of the tongue is due chiefly to lack of saliva and a dry tongue is usually furred A furred tongue may be found in the early morning in otherwise healthy persons in heavy smokers and in mouth breathers It may also be associated with digestive disturbances fevers or any long illness The tongue of scarlet fever at first shows bright red papillæ standing out of a thick white fur Later the white coat disappears leaving enlarged papillæ on a bright red surface—the strawberry tongue A brown fur on the tongue is due to a fungus infection of the papillæ and is of no special significance The amount of information to be gained from the presence or absence of fur on the tongue has been much exaggerated (4) The character of the papillæ Generalized atrophy of the papillæ produces a smooth or bald tongue which is characteristic of pernicious anæmia but may also sometimes be found in iron deficiency anæmia sprue other gastro intestinal disorders and deficiency states especially pellagra In severe cases smoothness may be associated with wrinkling of the mucous membrane which has then

to be distinguished from fissuring of the tongue seen in chronic superficial glossitis due to syphilis and congenital fissuring of the tongue which is of no pathological significance In chronic superficial glossitis areas of leukoplakia—whitish opaque areas of thickened epithelium—are separated by intervening smooth and scarred areas and no normal papillæ are seen In congenital fissuring (Plate 7) the papillæ are normal but the surface is interrupted by numerous irregular but more or less symmetrical furrows (5) The under surface of the tongue—a small ulcer on the frænum is sometimes seen in persistent coughing and particularly in whooping-cough Lastly (6) observe the edges of the tongue Look for ulcers indentations of the teeth areas of leukoplakia and smooth patches (Plate 7)

Inspect the mucous membrane on the inside of the cheeks In the catarrhal stage of measles before the appearance of the rash small bluish white spots surrounded by a red areola may be seen opposite the molar teeth These are known as Koplik s spots In the same situation irregular areas of slaty grey pigmentation are seen in Addison s disease

Thrush may sometimes be seen on the surface of the buccal mucous membrane especially in children It presents the appearance of small white points or patches raised somewhat above the surrounding surface which is sometimes redder than normal Patches of thrush are apt to be mistaken for small milk curds but curds can be easily detached while thrush patches can only be removed with difficulty and when removed tend to leave behind a raw surface To search for the fungus (*Saccharomyces albicans*) a small piece of the patch should be scraped off and examined in a drop of glycerin A quantity of epithelial debris along with bacteria and leucocytes will be seen and mixed up with these the filaments of the fungus These consist of long but unequal segments usually possessing a refractile nucleus at each end

The palate fauces and pharynx —Introduce a tongue depressor and note first the general colour of the soft palate fauces and pharynx observe any abnormal degree of pallor or redness Remember that great insensitivity of the palate and pharynx is common in hysterical patients Again look for any ulcers or mucous patches on the palate fauces or tonsils Mucous patches are slightly raised round or oval areas covered by pearly grey membrane They are found

in secondary syphilis as are also superficial circinate snail track ulcers Deep clean cut ulcers with sharp edges occur in Vincent s angina Large ragged ulcers or sloughs are seen in agranulocytosis and leukæmia Instruct the patient to make the sound Ah thus raising the soft palate and increasing visibility Look carefully at the **tonsils** noting any enlargement Yellowish or greyish points or patches may sometimes be seen on their surface See if these can be wiped off leaving a sound surface as is the case with accumulated follicular secretion or whether removal leaves a raw surface as happens with the false membrane of diphtheria Note always if the soft palate and uvula show any similar spots or patches The membrane of diphtheria is found characteristically on the mucous membrane of the fauces as well as on the tonsils Bacteriological examination of a throat swab (p 407) is essential when there is a suspicion of diphtheria Next look at the **pharynx** The presence upon its surface of a number of flat adenoid swellings somewhat like sago grains is so common as to be almost a normal appearance In chronic pharyngitis these are much increased A few dilated venules can also be frequently observed Notice any pus or excess of mucus on the surface and the existence of any ulceration In retropharyngeal abscess the posterior wall of the pharynx is bulged inwards Sometimes this can be more easily made out by palpation

The breath —Bad teeth gum or mucous membrane ulceration and retention and decomposition of secretion in the follicles of enlarged tonsils are the commonest sources of offensiveness in the mouth In gangrene of the lung the breath often has a putrid smell resembling that of the sputum In bronchiectasis also it sometimes has a peculiarly offensive odour that has been compared to sweet apple blossom with an *arrière gout* of stale fæces Fetor due to pulmonary conditions is elicited by asking the patient to cough Slighter degrees of offensiveness may be due to gastric disorder

In uræmia the breath may have a fishy or ammoniacal smell In diabetic coma the odour of acetone may be present Various drugs—e g paraldehyde—impart characteristic odours to the breath while patients who are taking bismuth sometimes produce a garlicky odour Iodides produce a peculiar fetor

THE ŒSOPHAGUS

Special anatomy —The œsophagus is from 9 in to 10 in long It begins opposite the cricoid cartilage and ends opposite the 9th thoracic spine It is crossed by the left bronchus between the 4th and 5th thoracic vertebræ

In cases of difficulty in swallowing the radiologist must be asked to screen the patient and to report upon the course down the œsophagus of radio opaque emulsion

II THE ABDOMEN

Anatomy —The natural lines on the surface of the abdomen are (1) the linea alba (2) the lineæ semilunares (3) the lineæ trans versæ

The structures lying behind the linea alba from above down wards are (*a*) the left lobe of the liver extending to about three fingers breadth below the ensiform (*b*) part of the stomach unless empty (*c*) the transverse colon (*d*) coils of intestine covered by omentum (*e*) the bladder when distended and the uterus when pregnant

The linea semilunaris corresponds to the lateral border of the rectus abdominis muscle

The lineæ transversæ are the three fibrous intersections in the upper part of the rectus abdominis muscle

The abdomen has been artificially divided into regions by means of vertical and horizontal lines The vertical lines are drawn upwards from the mid point of the inguinal ligament on each side The transverse lines are (1) the subcostal or infracostal drawn across horizontally at the level of the lowest points of the 10th costal arches and (2) the intertubercular or bi iliac between the tubercles marking the most prominent points of each iliac crest Nine regions are thus marked off in three vertical rows Those in the middle row are from above downwards the epi gastric umbilical and hypogastric and in each lateral row we have the (right or left) hypochondriac lumbar and iliac regions

The *umbilicus* lies opposite the upper part of the 4th lumbar vertebra Its position is far too variable for it to be a trustworthy landmark

The *aorta* bifurcates about ¾ in below and slightly to the left of the umbilicus *the iliac arteries running in a line drawn from*

that point to a point midway between the anterior superior spine and the symphysis pubis

The *cœliac axis* arises at a point 4½ in to 5 in above the umbilicus and the *renal arteries* about an inch lower than the cœliac axis

The *transpyloric plane* is often used as a guide in the examination of the abdomen It is defined as lying midway between the supra sternal notch and the upper border of the symphysis pubis It usually lies about halfway between the xiphisternal junction and the umbilicus and it corresponds posteriorly with the lower border of the 1st lumbar vertebra

GENERAL EXAMINATION OF THE ABDOMEN

The patient should be lying flat on his back in a good light The abdomen is exposed by turning down all the bedclothes except the inner sheet The clothing should then be drawn up and lastly the sheet folded down a little above the level of the pubes These details are of special importance in examining female patients Before beginning the examination of the abdomen ensure that the bladder is empty Flexion of the hips is rarely of help Better relaxation is sometimes obtained in a semi recumbent position but access to the abdomen is naturally less since the costal margin is then made to approximate to the pelvis Nearly always the abdomen is best examined with the patient fully recumbent

Inspection of abdomen —First look at the general contour of the abdomen Is it of normal fullness is it swollen or protuberant or is it sunken or retracted ? If there is any bulging note if it is general or local A remark was once made that general fullness may be due to fat fluid flatus or fæces to which aphorism may be added fœtus in the case of women A *new growth* may also be a cause of general abdominal tumidity In general bulging it should be noted whether the distension is most marked in the antero posterior or in the transverse diameter In cases of general abdominal swelling measurement should be made at either the umbilical level or at the point of maximum distension Observe in which zone local bulging is situated Lastly note if there is any movement to be seen in the swelling either along with or independently of respiration

Pulsation in the epigastric region may be noticed on abdominal

inspection The causes of it are in order of frequency —(1) Aortic pulsation which may be visible in any slightly built patient with a thin abdominal wall (2) Transmitted pulsation from a tumour (often a carcinoma of the stomach) overlying the aorta (3) Distension of the right ventricle (4) Venous pulsation of the liver (5) Aneurysmal the pulsation in this case is expansile

The movements of the abdominal walls should be studied Normally they bulge during inspiration and fall in during expiration *Paralysis of the diaphragm produces the opposite picture* sometimes the paralysis is unilateral in which case one side of the abdomen will move naturally Absence of movement of the abdominal walls is a valuable sign of peritonitis

Sometimes peristaltic waves are visible through the abdominal wall This is often the case in chronic intestinal obstruction The coils of intestine above the constricted part stand out prominently A definite pattern of abdominal tumidity results depending on the site of the obstruction If there is a constriction at the ileo-cæcal valve the distended coils of small intestine tend to stand out in the centre of the abdomen one above the other so as to form a ladder pattern If the obstruction is low down say in the sigmoid flexure the periphery of the abdomen is chiefly affected A dilated stomach may also stand out as a prominent tumour in which peristaltic waves are visible Peristaltic waves in the stomach run from left to right

Small intestinal peristalsis is sometimes seen in elderly people with thin abdominal walls especially in the presence of divarication of the rectus abdominis muscles in the absence of any abdominal disease With this exception it is almost always a sign of intestinal obstruction of some kind It is thus an important sign and in order not to miss it it may be necessary to sit and observe the abdomen carefully for several minutes In the case of congenital pyloric stenosis of infants visible peristalsis may be the only diagnostic sign and the minutes thus spent are never wasted

Attention should next be paid to the surface of the abdomen In great distension the surface is smooth and glossy *Striæ* (white or lilac lines in the epidermis) should be sought they indicate a recent change in the size of the abdomen and thus are found in obesity pregnancy ascites and wasting diseases Conspicuous purplish striæ are characteristic of Cushing s basophilism syndrome

Note any *distension of the surface veins* and endeavour to ascertain in what direction their blood is flowing In obstruction of the inferior vena cava the inferior epigastric veins are full from the establishment of a collateral circulation In such cases also a large *lateral* vein can be seen running up about the midaxillary line establishing a communication with the tributaries of the superior vena cava In portal obstruction a number of distended veins may rarely be seen radiating from the umbilicus They may be brought out only on coughing To this appearance the term caput Medusæ has been applied It is due to establishment of a connexion between the portal and parietal veins by means of the round ligament *Pigmentation* of the abdominal wall is sometimes important Along the middle line it forms the linea nigra—one of the signs of pregnancy Note the appearance of the *umbilicus* Is it depressed level with the surface bulging or everted as in gross ascites? Lastly one should never omit to look at the usual sites for any evidence of hernia

Palpation of the abdomen —The patient should lie flat on his back He should be told to keep the mouth open and to breathe quietly his attention may be diverted by conversation The observer must sit or kneel beside the patient in order to get his hand into the horizontal position Ordinary palpation is performed with one hand only The hand must be warm In order to gain the confidence of the patient the hand should be allowed to rest for a moment on the surface of the abdomen before palpation is actually begun Each region should be palpated systematically Poking with the finger tips should be avoided the best movement being a gentle one from the metacarpo phalangeal joints During expiration the receding abdominal wall should be followed by the fingers and a gentle rotatory motion of the finger tips may then be carried out This often enables one to feel the deeper structures better than by simple pressure To examine lateral regions of the abdomen bimanual palpation is carried out The physician sits or kneels by the bedside One hand is placed posteriorly in the interspace between the last rib and the crest of the ilium The other is placed over the abdominal wall in front The posterior wall is then pushed up against the hand in front so that any structure lying between the two hands can be distinctly felt The secret of the method consists in keeping the front hand as still as

possible This procedure is of special value in the examination of the kidneys

First notice the degree of tension of the walls and *resistance* experienced Begin always by a systematic very light palpation of the whole abdomen noting any local or general resistance or any marked tenderness In this way the patient s confidence is gained and the later deep palpation rendered easier Normally the abdomen has an elastic or doughy feeling only to be learnt by experience In disease the resistance may be increased It should be observed whether this increase is general or local General peritonitis produces a great increase in the resistance from a reflex contraction of the muscles of the abdominal wall Local increase in resistance is frequently due to localized peritonitis and is of great diagnostic value Palpation of the normal abdomen is painless If tenderness is elicited its exact extent and point of maximum intensity should be noted Tumours should be carefully felt for and care taken that a bulging rectus muscle does not cause confusion The outline of the rectus muscle will become obvious if the patient is asked to raise his head against resistance

If a tumour is really present, determine whether it is situated inside the abdomen or in the abdominal wall Try to move the abdominal wall from side to side over the tumour If the growth is intra abdominal this can usually be done without difficulty unless it has become adherent to the parietal peritoneum Try also to grasp the tumour and to make the fingers meet as it were under it This can usually be accomplished in the case of tumours situated wholly in the abdominal wal'

If the tumour is intra abdominal it must be decided where it is growing from and especially whether it is coming up out of the pelvis or is truly abdominal To decide this place the hand about one inch below the umbilicus and push backwards and downwards in the *direction of the sacral promontory One can then feel* whether the tumour is passing down into the pelvis or not The size and shape of the tumour should be noted and the nature of its surface—whether smooth or nodular The presence or absence of fluctuation should then be investigated

The *mobility* of a tumour is a most important point The directions in which a tumour can be moved should be noted and whether movement is influenced by respiration This is a point of special value Tumours connected with the liver and spleen move

freely with respiration and so may those of the stomach Tumours of the kidney may be slightly movable Those connected with the other abdominal organs do not move with respiration at all unless they have contracted adhesions

Percussion of the abdomen—This should be carried out in the same manner as will be described for the chest but particular care should be taken to percuss *lightly* Percussion of the normal abdomen yields a tympanitic note throughout except in the regions of liver and splenic dullness or over a full bladder Enlargement of these organs may sometimes be confirmed by percussion The liver dullness is absent when there is gas or air in the peritoneal cavity Such absence is a sign of perforation of a viscus usually of perforation of a gastric or duodenal ulcer but may be due to a therapeutic pneumo peritoneum

Free fluid in the peritoneum (*ascites*) is distinguished by the fact that it shifts its position with that of the patient If he is turned over on his side and time given for the intestines to float up the uppermost flank becomes resonant while the height of the dullness on the lower side rises This phenomenon is known as shifting dullness

The *fluid thrill* is another physical sign of fluid in the peritoneum To elicit this sign the patient is laid on his back one hand is placed over the lumbar region on one side and the opposite side is flicked or tapped with the fingers of the other hand A distinct impact will be felt to pass from one hand to the other A similar impulse can be transmitted through a fat abdominal wall and so it is necessary for an assistant to place the edge of his hand *firmly in the middle line of the abdomen while the tapping is* performed This damps down vibrations transmitted by the wall On the whole the results of simple percussion afford the best evidence of the presence of ascites A fluid thrill can only be expected when the amount of fluid is large and under tension

Fat is distinguished by taking the abdominal wall between the hands and pinching it up *gas* by the results of percussion Of *new growths* ovarian cyst is probably the most liable to be mistaken for ascites An ovarian tumour however causes an antero-posterior bulging of the abdomen while in ascites the bulging is mainly lateral In ovarian tumours the dullness is central and does not change with the position of the patient in ascites the

chief dullness is in the flanks and it shifts when the patient is moved in ascites the umbilicus is flat or bulges out while in ovarian tumours it is drawn upwards and the slit in the umbilicus is usually transverse in ascites and vertical in ovarian tumour

Auscultation of the abdomen —Normally on auscultation of the abdomen numerous borborygmi are audible However in cases of general peritonitis or of intestinal ileus these sounds may be entirely absent and the silent abdomen is a valuable physical sign

III THE ABDOMINAL VISCERA

THE STOMACH

Special anatomy (Plates 8 11) —The normal stomach in the living subject is shaped like the letter J The cardiac orifice usually lies on the left side of the 11th thoracic vertebra and 4 in behind the 7th left costal cartilage 1 in from the left border of the sternum The position of the pylorus is at or just to the right of the mid line and midway between the infrasternal notch and the umbilicus It is normally under cover of the liver The fundus of the stomach may reach as high as the 5th interspace in the midclavicular line and rise a little above and behind the apex beat of the heart Only a small part of the body of the stomach and of the pyloric region is in contact with the anterior abdominal wall The exact position of the greater curvature varies greatly according to the degree of distension of the stomach and the posture of the patient radio graphic examination has shown that it may be found anywhere from the region of the epigastrium and left hypochondrium to well below the brim of the pelvis and so the outline of the stomach in the cadaver shown in Plate 8 must therefore be regarded as diagrammatic

Inspection of the stomach region is included in the general examina tion of the abdomen (p 39)

Palpation of the stomach —Note any *tenderness* and define the point of its greatest intensity Examine for *tumours* The com monest tumour is a carcinoma of the pylorus Lastly try for *splashing* To elicit this sit at the left side of the patient with one hand over the left lower ribs behind with the other placed over the

front of the stomach make short sudden dipping movements Splashing is partly heard and partly felt

Distinct splashing elicited three hours after a meal especially below the level of the umbilicus is suggestive of a dilated stomach but a splash may be elicited over a normal stomach shortly after a meal containing much fluid especially if the abdominal wall is thin Care should be taken not to mistake a splash produced in the transverse colon for a stomach splash

THE LIVER

Special anatomy (Plates 8 10) —The liver lies chiefly in the right hypochondrium Its left lobe extends across the epigastric region but does not pass more than 2 in to the left of the sternum Above the liver reaches almost to the nipple below it extends to the costal margin The lower border passes obliquely upwards from *the 9th right to the 8th left costal cartilage* crossing the mid line somewhat above the mid point between the base of the xiphoid and the umbilicus

The gall bladder is situated just internally to the 9th right costal cartilage and immediately to the outer side of the right rectus muscle

Inspection of the liver is of little value Any visible swelling fullness pulsation or œdema of the overlying skin should be noted The edge of the liver can sometimes be seen when the organ is enlarged It forms a sharp line which moves with respiration

Palpation of the liver —Feel for the lower edge To do this place the hand flat on the abdomen its edge towards the costal margin and to the outer side of the rectus muscle thus avoiding the upper septum of the rectus sheath which may be mistaken for the lower edge of the liver Depress the edge of the hand slightly so as to push up a fold of skin and ask the patient to take a long breath If the edge of the liver is palpable it will be felt to ride under the edge of the hand Trial must be made at different levels before it is decided that the edge cannot be felt The edge of the liver can only rarely be felt in health It moves down with inspiration The character of the edge should be noted—whether it is smooth or irregular thickened or sharp

The surface of the liver in the epigastrium should then be felt in the usual way Any tenderness should be noted and whether it is localized or general The character of the surface should be made out whether it is soft smooth and tender as in heart failure firm and finely nodular as in portal cirrhosis or hard and coarsely irregular as in secondary carcinoma Be careful not to confound little irregularities which are frequently present in the upper parts *of the recti with irregularities on the surface of the liver*

Percussion of the liver —The patient should be lying down for percussion of the anterior and lateral aspects sitting up or standing for the posterior aspect

Use fairly heavy percussion Begin high up at about the 2nd rib to get a good lung note and percuss down until impairment is detected The upper limit of liver dullness forms an almost horizontal line around the chest To define the lower edge of the liver use very light percussion and pass upwards

The gall bladder is examined by palpation and percussion It *cannot be felt unless distended when it may form a smooth pear* shaped swelling situated just to the outer edge of the right rectus muscle It can be moved freely from side to side round a point opposite to the 9th costal cartilage It moves with respiration

If present *tenderness* of the gall bladder can be elicited by placing the hand beneath the costal margin in the right hypo chondrium The patient is told to take a deep breath and at the same time the hand is moved upwards underneath the costal margin As the diaphragm descends the gall bladder is driven against the fingers and if it is tender the breath is at once arrested with a gasp This is spoken of as Murphy s sign

On percussion a distended gall bladder forms a dull area pro jecting from the liver dullness towards the umbilicus but usually continuous with it

THE SPLEEN

Special anatomy (Plates 8 10) —The spleen lies in the left hypo-chondrium It is bounded above and laterally by lung medially by stomach and intestine Its lower end rests upon the phrenico colic fold of peritoneum It lies along the 9th 10th and 11th ribs being partially separated from them by the diaphragm and lower edge of the left lung Its upper end is opposite the 9th thoracic

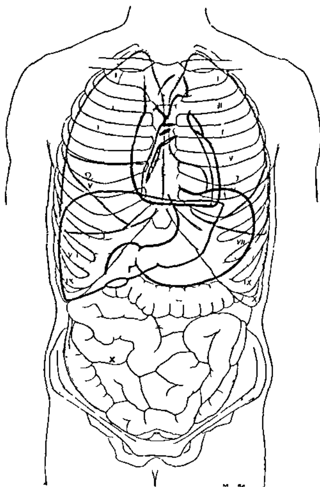


Plate 8 —VISCERA OF THORAX AND ABDOMEN AS SEEN FROM THE FRONT IN THE CADAVER Scale 1 = 5 6

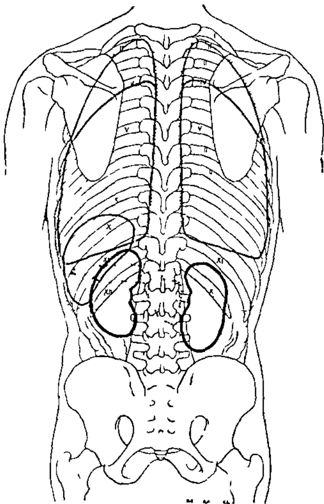


Plate 9.—Viscera of Thorax and Abdomen as seen from Behind in the Cadaver. Scale 1 = 5 6

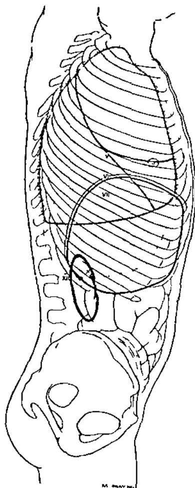


Plate 10.—VISCERA OF THORAX AND ABDOMEN AS SEEN FROM THE RIGHT SIDE IN THE CADAVER Sc 1 1—56

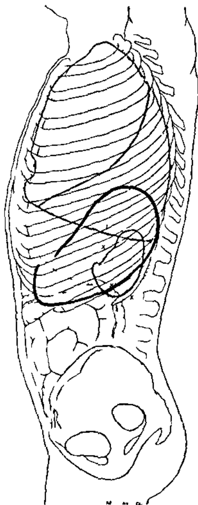


Plate 11 —Viscera of Thorax and Abdomen as seen from the Left Side in the Cadaver Sc 1 1 = 5 6

spine and is about 1½ in from the [illegible] line. Its [illegible] as far forward as the midaxillary line

Inspection of the spleen —If much en[illegible], the spleen [illegible] a visible tumour in the left side of the abdomen which [illegible] with respiration.

Palpation of the spleen —This is the [illegible] important method of investigating the spleen. If [illegible] a palpable spleen is enlarged. It is never safe to [illegible] enlargement of the spleen unless it is palpable.

Feel for the spleen by [illegible] to the right side of the patient. Place the flat of the left hand over the edge of the costal margin at the left flank and firmly bring the ribs and lateral abdominal wall medially as the patient breath[illegible] in. Then start palpating with the right hand well down towards the right iliac fossa and work up towards the costal margin. (Large spleens are missed by starting palpation too near the costal margin.) The edge of the enlarged organ will be felt against the fingers of the right hand. Sometimes in the case of minor degrees of splenomegaly the organ is best felt if the patient rolls over half on to his right side towards the examiner.

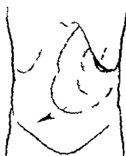


Fig. 5. —Diagram to show direction of enlargement of the spleen

The edge of the spleen is sharp and usually quite smooth. Notches can often be felt in it [illegible]. It is important to note that the spleen enlarges [illegible] and to the right towards the right iliac fossa (Fig. 5) [illegible] border of an enlarged spleen is always directed [illegible] inwards.

Auscultation over the spleen may be [illegible] to detect the existence of friction. This [illegible] and over the surface of splenic infarcts

THE KIDNEYS

Special anatomy (Figs. 9, 10). [illegible] lies partly in the epigastric, partly in the hy[illegible] region. The right kidney

lies partly in the lumbar region as well The kidneys are higher in relation to the anterior abdominal wall than is sometimes thought The lower end of the right kidney is 1 in above the umbilicus the left about ½ in higher The lower end of each is about 3 in from the middle line

Posteriorly about one third of each kidney lies above the last rib The upper end of the right kidney is at the level of the 11th thoracic spine whilst its lower end is about 1 in above the iliac crest The left kidney is about ½ in higher

Palpation of the kidneys —The patient must be on his back The lumbar region must be flat and not arched forward Sit or kneel beside the patient Place one hand upon and below the last rib *behind the other immediately below the costal margin in front* The posterior hand should press the loin forwards while the other hand pushes the anterior abdominal wall backwards upwards and inwards The kidney will then be felt between the two hands if it is at all enlarged or displaced Even in health (if the patient is not too fat) the lower part of the organ can often be felt

If the kidney is not felt in this way ask the patient to take a long sighing inspiration If the front hand moves upwards and inwards as the patient inspires the kidney may be caught between the two hands

The kidney moves slightly with respiration Exaggerated mobility indicates *movable kidney* Ease of palpation of the kidneys varies greatly with the build of the patient In health the lower pole of the right kidney is frequently palpable in persons of spare build the left seldom so

A movable right kidney is apt to be mistaken for a distended gall bladder and vice versa The shape size and consistence of the tumour may be apparently identical in the two cases One point of distinction is that a distended gall bladder can be temporarily pushed back from the abdominal wall but always tends to spring forward again it is therefore always in evidence A floating kidney however often disappears for a time and can only with difficulty be grasped again Another point of distinction is that a kidney can be pushed down towards the pelvis and held there during even forcible expiration whilst the gall bladder moves upwards again during the expiratory act

An enlarged left kidney may be mistaken for the spleen

points of distinction are (1) The spleen has a sharp edge in which a notch can often be felt The edge of the kidney is *always* rounded and has no notch (2) The fingers can usually be passed between the upper end of a kidney tumour and the ribs but not between the ribs and a splenic tumour (3) The colonic resonance may be detected passing in front of a renal tumour but not so in the case of a splenic tumour (4) A renal tumour is usually bimanually palpable that is can be moved backwards and forwards between one hand in the loin behind and the other on the anterior abdominal wall An enlarged spleen is not bimanually palpable

An enlarged kidney tends to bulge forwards Perinephric abscesses etc bulge backwards

THE INTESTINES

Special anatomy —The ileum joins the colon at a point 2 in internal to and somewhat above the right anterior superior iliac spine The apex of the cæcum corresponds to a point a little to the inner side of the middle of the inguinal ligament The base of the vermiform appendix usually lies opposite a point 1½ to 2 in from the anterior superior spine along a line drawn from that spine to the umbilicus This is sometimes called McBurney s point because he showed that in the majority of cases of appendicitis it is the point of maximum tenderness

The splenic flexure of the colon lies behind the stomach the hepatic lies under cover of the liver The former is at a higher level than the latter The transverse colon passes across the abdomen in a curved direction Radiography has shown that its position is very variable

Examination of the intestines by **inspection and palpation** has been described under the general examination of the abdomen

Rectal examination —Place the patient in a good light and in a semi prone position—i e resting on the left breast with the right thigh and knee well drawn up the inner aspect of the right knee resting on the couch Draw aside the buttocks and inspect the region of the anus noting the presence of any eruption of external hæmorrhoids etc Fit a finger stall to the right forefinger and smear it with petroleum jelly If no finger stall is available fill the nail with soap and smear the finger with petroleum jelly Massage the anus for a moment with the finger and then pass it gently

through the anus directing it slightly forwards at first Note the degree of resistance offered by the sphincter this shows whether the latter is normal spasmodic or relaxed

Once the anal canal is passed direct the finger slightly backwards and upwards asking the patient to bear down a little at the same time The finger can then be swept round and the whole inner surface of the rectum explored

In the male the prostate will be felt projecting into the rectum and above it the trigone of the bladder flanked by the seminal vesicles below is the membraneous urethra In the female the cervix will be felt projecting back in the form of a firm rounded swelling Feel the mucous membrane for polypi ulcers and malignant neoplasm

Remember that hæmorrhoids are not palpable unless they are thrombosed The presence of scybala or foreign bodies can be determined If the lymphatic glands which lie in the hollow of the sacrum are enlarged they may be felt If secondary malignant deposits or an abscess be present in the recto vesical pouch the mass will be palpable through the wall of the rectum On withdrawing the finger examine the finger stall for the presence of mucus blood or melæna

Proctoscopy and sigmoidoscopy —If rectal examination is negative and there is reason to suspect abnormality near the anus the anal canal and lower three inches of the rectum should be examined with the proctoscope Place the patient in the position described for rectal examination and pass the warmed lubricated instrument carefully to its full depth Remove the obturator and inspect the mucous membrane as the instrument is slowly withdrawn In this manner hæmorrhoids may be seen or the nature of a palpable abnormality directly ascertained

It is often necessary to examine the rectum and colon more fully than is possible by proctoscopy and in such cases the sigmoidoscope is employed Sigmoidoscopy requires skill and experience In accomplished hands the instrument can be passed for at least 20 cm and a further 4 cm or so of the colon is visible beyond this The procedure causes very little discomfort and anæsthesia is unnecessary and undesirable

Sigmoidoscopy is particularly useful in the differential diagnosis of diarrhœa of colonic origin It serves to distinguish carcinoma

of the colon the ulcerated bleeding mucosa of ulcerative colitis the polypi in polyposis coli and the red granular surface in granular proctitis In suspected amœbic dysentery the mucous membrane may be inspected and portions of mucus or scrapings from ulcers may be removed mounted in saline on slides and examined microscopically for amœbæ and cysts When the sigmoidoscope is used for this purpose it should be lubricated with a non greasy preparation such as mucilage or droplets of oil will interfere with the microscopical examination

IV SPECIAL INVESTIGATIONS OF THE ALIMENTARY AND BILIARY TRACTS

Under this heading methods of examining the stomach gall bladder and intestine by intubation and radiology will be described

1 THE FRACTIONAL TEST MEAL

The apparatus required is a narrow bore stomach tube a 20 ml Record syringe and a rack of twelve numbered test tubes The most suitable stomach tube is that devised by Ryle (Fig 6) It is of thin rubber of about 8 mm external circumference and is marked by one transverse line of 40 cm to indicate the teeth-cardiac

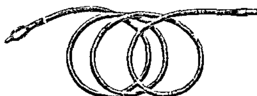


Fig 6 —Ryle s stomach tube

orifice distance and by three transverse lines at 57 cm to indicate the teeth pylorus distance It has a mall blind bulbous extremity weighted with metal and at a distance 2 cm from the tip is perforated by a number of small holes 2 mm in diameter The resilient edges of these perforations obviate the risk of damage to the gastric mucosa when strong suction is exerted on an almost empty stomach

The test breakfast consists of thin gruel made by adding a quart of water to two tablespoonfuls of fine oatmeal boiling down slowly to a pint and straining through muslin The patient is given a light supper all drugs are suspended and he is starved from mid night At 9 a m the tube is boiled and placed in warm water whilst the patient sits up in bed or in an armchair a towel being arranged across his chest The tube is not lubricated but is placed on the back of the patient s tongue and he is asked to close his mouth and to swallow the bulbous end just like a pill Swallowing is continued until the pyloric mark almost reaches the teeth the patient meanwhile being reassured and told to breathe gently through his nose

A sample of about 15 ml of the fasting stomach contents is withdrawn by gentle aspiration with the syringe though sometimes the whole of the resting fluid is withdrawn and measured Without withdrawing the tube the patient drinks the pint of warm gruel after which at fifteen minute intervals samples of stomach content (10–15 ml) are aspirated the tube remaining *in situ* for three hours

After the withdrawal of each specimen air is injected to empty the tube and a small stopper used to close the end of the tube thus preventing leakage Blocking of the tube is easily overcome by air pressure In most cases there is no difficulty in obtaining twelve specimens each of 15 ml With marked hyposecretion difficulty may be encountered in the withdrawal of specimens and each may measure only 3 ml If difficulty is experienced in with drawing the specimens the tube should be withdrawn or swallowed a few centimetres or the patient may be tilted well towards the left At the end of three hours the tube is gently withdrawn the patient being asked to swallow as it passes the level of the cricoid

In the laboratory the naked-eye appearance of each specimen and the amount and colour of both sediment and supernatant fluid are noted A rough estimate of the amount of each specimen is made and the presence or absence of bile blood and mucus is noted Every specimen is then examined for free HCl and total acidity by the method described below The emptying time of the stomach is estimated by the addition of a few drops of iodine solution to each tube the presence of starch being shown by an intense blue coloration The curves of free HCl and total acidity are then plotted as shown in Figs 7 and 8 the first reading being that of the fasting gastric content The rate of emptying of the

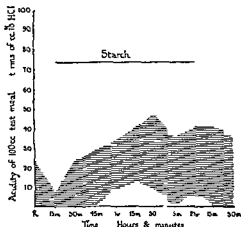


Fig 7 —Normal gastric function

F m bserv t n by B nnett d Ryl n 100 h lthy mal s.
(G y H p R pt 1921 l x 317)

Th h d d a e p esent the *l m t f f* *HCl* in 80 p r c t f h althy m l

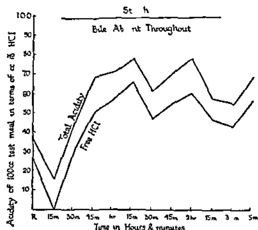


Fig 8 —Fractional test meal chronic ulcer of duodenum

Sp um w thdrawn w th All dep t ry lean. Clea col url ss supern tan
fl d No m s. Fl d lik wat in la t three pecimens.

stomach and the presence of bile blood and mucus are plotted horizontally as black lines

Estimation of total acidity and free HCl—Take 10 ml of the filtered gastric contents (in a beaker) add a few drops of Topfer s reagent, and cautiously run in decinormal caustic soda solution until the pink colour is discharged Read the burette (first reading) Now add a few drops of an alcoholic solution of phenol phthalein Again run in the soda solution this time until the least trace of persistent pink colour appears in the beaker This can best be appreciated by holding against a white surface Read the burette (second reading) The first reading gives the amount of the free acidity the second reading shows both the total and the combined acidity the total by the number of cubic centimetres run in from the beginning of the titration the combined by the number added after the first reading was made The term combined as u ed here includes acid combined with proteins with enzymes and with mineral bases in the form of acid salts Acidity due to free organic acids will also be included in the second reading of the burette

Should there be no free acid, the deficit " may be determined i e $\frac{N}{10}$HCl is added to 10 ml of the filtered contents until free acid is present Then after adding phenol phthalein the acidity is determined with $\frac{N}{10}$NaOH In this case the total acidity is obviously the number of cubic centimetres of alkali used minus the number of cubic centimetres of $\frac{N}{10}$ acid previously added

The results of these titrations may be stated in one of two ways

(1) Directly from the number of cubic centimetres of $\frac{N}{10}$NaOH required to neutralize 100 ml of the stomach contents e g if to neutralize the 10 ml of contents 5 c c of $\frac{N}{10}$NaOH were necessary then for 100 ml of contents 50 ml of soda would be required and the total acidity would be 50 If of this 5 ml of soda 3 ml were required to neutralize the free acid and 2 ml to neutralize the combined acid the " free and combined acidities would be respectively 30 and 20 Normally the total acidity varies from 40 to 70

(2) Indirectly in terms of HCl Thus one litre of decinormal soda is required to neutralize 3 65 grm of HCl If therefore 100 ml of stomach contents require 50 ml of soda to neutralize them then the acidity of the 100 ml is equal to that of 0 18 grm HCl—that is to say

the acidity is 0 18 per cent The normal total acidity in terms of HCl is about 0 2 per cent The necessity for calculation may be avoided if one remembers that provided 10 ml of contents have been taken and that decinormal soda is used for titration the number of ml of soda required × 0 365 = HCl per 1 000 parts In order to get the percentage of HCl one has merely to shift the decimal point one place to the left For example if 10 ml of decinormal soda solution were required to neutralize 10 ml of gastric contents the amount of HCl present is 3 65 per 1 000 or 0 365 p r cent

The fractional test meal is employed to assist in the diagnosis of peptic ulcer gastritis pyloric obstruction carcinoma of the stomach and pernicious anæmia The findings should be noted under the following headings

(1) The amount and character of the resting fluid

(2) The presence in the specimens of fresh or altered blood excessive mucus or food taken on the previous day

(3) The rate of emptying, as indicated by the persistence of starch in the samples and the occurrence of regurgitation from the duodenum as indicated by the presence of bile

(4) The presence or absence of free HCl and the character of the curves for total acidity and free HCl

Fig 7 shows the limits of normal free HCl in 80 per cent of 100 healthy males (young medical students) The incidence of achlorhydria in otherwise normal persons increases considerably with age

The normal amount of resting fluid varies from 10 to 150 ml A very large excess containing remains of food from the previous day indicates pyloric obstruction The presence of mucus in excessive amounts suggests gastritis A little fresh blood may be due to trauma but the presence of altered blood or the persistence of blood in many specimens suggests ulcer or carcinoma usually the latter (Fig 9) A resting fluid of large volume with altered blood and a foul smell strongly suggests carcinoma The usual emptying time is from 1¼ to 2½ hours Rapid emptying indicates hypermotility Slow emptying with a climbing acid curve indicates pylorospasm or pyloric obstruction A high acid curve is common in duodenal or pyloric ulcers (Fig 8) but may be found in their absence Achlorhydria is constantly found in true pernicious anæmia (Fig 10) and is characteristic of gastric carcinoma (Fig 9) but may be found in a proportion of healthy persons in various other diseases and occasionally in patients with gastric ulcers

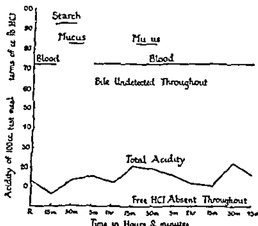


Fig 9 —Fractional test meal carcinoma of stomach

Spec m n w thdr w w th e All dirty and t rb d a d m jority sta ned b sht d w th blood P rridg v ble p to 30 m ut s Floating bl od t n d m s most pecum ns

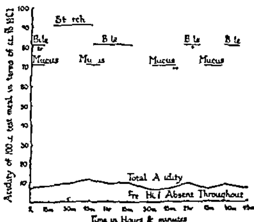


Fig 10 —Fractional test meal pernicious anæmia

Specimens withd awn with great d fficulty mo t f th m being o ly 5 ml Much mucus. Porridge v ble to o e ho Th n d rty t rb d fl d.

Conversely though achlorhydria is characteristic of carcinoma this disease is occasionally found in the presence of free HCl

The fractional test meal therefore though of some confirmatory value has not proved as useful as was at first hoped In the opinion of many physicians its principal value is in confirming the diagnosis of pernicious anæmia and for that purpose the histamine test meal should be used

2 THE HISTAMINE TEST MEAL

There is no standard method of carrying out this test but for most purposes it is convenient to combine it with the fractional test meal This is done in the following manner

The patient is prepared for and given a gruel meal after swallowing a Ryle s stomach tube as described previously Specimens are withdrawn at fifteen minute intervals up to one hour At this stage the specimen withdrawn is tested by means of Congo red paper which turns blue if free acid is present If it is not present an injection of 0 5 mg of histamine acid phosphate is administered subcutaneously and further specimens are withdrawn for a further two hours The blood pressure should be taken beforehand and if low the dose of histamine should be reduced to 0 25 mg Analysis of the specimens is carried out in a manner similar to that already described for the gruel test meal Following the injection of histamine most patients experience slight palpitation flushing of the skin and headache but these symptoms are transitory

Histamine fast achlorhydria is usually associated with an absence or greatly diminished production of pepsin and of intrinsic factor This condition is found constantly in true pernicious anæmia and is known as *achylia gastrica* If the Congo red test is positive for hydrochloric acid at one hour in the course of the fractional test meal then the absence of achylia is conclusively demonstrated and there is little object in administering histamine

3 DUODENAL INTUBATION

Samples of bile can be collected by duodenal intubation A Ryle s tube is introduced into the stomach in the manner already described and the stomach contents are withdrawn The patient then lies on the right side with his pelvis elevated on pillows Usually within an hour the bulb passes into the duodenum as indicated by the fact that the fluid withdrawn is alkaline to litmus

or consists of frank bile The position of the bulb is better confirmed radiologically A stimulant to the biliary reflex is then injected through the tube This may be 30 ml of 33 per cent magnesium sulphate 20 ml of 10 per cent peptone or 30 ml of olive oil warmed to body temperature before injection Air is then blown through the tube to empty it and the end clipped After a few minutes dark bile can usually be withdrawn readily If the bile is wanted for histological examination magnesium sulphate must not be used as it has been shown to promote a desquamation of epithelial cells that may easily be mistaken for pus cells

4 THE BARIUM MEAL FOLLOW THROUGH MEAL BARIUM ENEMA AND CHOLECYSTOGRAM

These radiological methods are often of the greatest value but for details of their performance the student must consult special textbooks

For the barium meal the patient swallows a paste or suspension of radiopaque barium sulphate while the radiologist observes its passage on the fluorescent screen Films may be taken to provide a permanent record of any abnormality discovered but as in all barium examinations of the alimentary tract it is the results of screening which are important The barium meal is principally used in the diagnosis of gastric and duodenal ulcer and of gastric carcinoma

In gastric ulcer the radiologist may demonstrate—

(i) an ulcer crater as indicated by a projection of barium or by a characteristic distortion of the mucosal pattern

(ii) a localized spasm of the stomach opposite the ulcer known as an incisura

(iii) changes indicating the gastritis which accompanies an ulcer including—

(a) an increase in the rugosity of the mucosal pattern
(b) changes in gastric motility such as hyperperistalsis and rapid emptying

In *duodenal ulcer*—

(i) an ulcer crater
(ii) deformities of the duodenal cap due to spasm or scarring—
(a) generalized spasm (trefoil deformity)
(b) localized spasm (Åkerlund deformity)

(iii) other changes associated with duodenal ulcer such as an increase in the amount of resting gastric juice rapid emptying of the stomach or evidence of pyloric obstruction

In *gastric carcinoma* a portion of the stomach may be found which cannot by any manipulation be filled with barium and which remains constant in outline It is known as a filling defect.

Evidence may also be found of complications such as hour glass constriction of the stomach due to ulcer or pyloric obstruction due to ulcer or carcinoma

For some purposes the progress of the barium is followed through the small and large intestines This constitutes the *follow through meal*

In the *barium enema* the suspension is introduced into the rectum as an enema and manipulated round the colon to the cæcum By this means obstruction to the colon neoplasm diverticulosis and other abnormalities can be recognized

Cholecystography depends on the fact that certain substances such as sodium tetraiodophenolphthalein (Opacol or Shadocol) and derivatives of phenyl propionic acid (Pheniodol) are excreted by the liver into the bile and so render the gall bladder opaque After a preliminary radiograph has been taken to see if radiopaque gall stones are present, a dose of the preparation suitable for the patient s weight is given by mouth after the patient s evening meal which should be light and free from fat Several radiographs are taken on the following day If a normal gall bladder is seen the patient is given a fatty meal and its emptying is observed

Failure to demonstrate the gall bladder may be due to the fact that the dye was vomited or not absorbed on account of diarrhœa or other cause that hepatic function is impaired—it is generally useless to attempt cholecystography in a jaundiced patient—or that disease of the gall bladder is present Deformities of the gall bladder shadow due to structural changes and calculi whether opaque or not to X rays may thus be demonstrated

5 GASTROSCOPY

Gastroscopy is a specialized method of investigation demanding considerable experience The technical procedure will not be described here By means of this instrument in a large majority of cases almost the whole of the stomach from cardia to pylorus may be inspected The procedure is safe provided that no

œsophageal abnormality is present It is carried out under local anæsthesia without any great disturbance to the patient Thus doubtful radiological appearances may be confirmed or refuted and it may be possible to express an opinion as to the malignancy or otherwise of a gastric ulcer seen on X ray examination The stages of healing of a simple gastric ulcer may be followed and it is usually found that an ulcer crater can be seen gastroscopically for a considerable time after radiological criteria have pronounced the ulcer healed An important contribution of the gastroscope to gastric diagnosis lies in the recognition of the various types of gastritis and a diagnosis of gastritis is almost impossible without the aid of this instrument

6 LIVER FUNCTION TESTS

The liver is remarkable for the number and variety of its functions Numerous tests of individual functions have been used No single test can give a composite picture of liver function The more important tests are —

(1) Tests depending on failure to excrete bilirubin
  - (a) **Plasma bilirubin** See p 169
  - (b) **Urobilinogen in urine** See p 227

(2) Tests depending on metabolism of protein
  - (a) **Plasma proteins** The liver is mainly if not wholly responsible for the production of serum albumen whereas globulin is mainly produced elsewhere In advanced liver disease particularly in cirrhosis there is a considerable reduction in plasma albumen (normal 4 0 to 5 5 grm per 100 ml) without a corresponding fall in globulin (normal 1 4 to 3 0 grm per 100 ml) which may even be increased and thus there is a fall in the albumen globulin ratio
  - (b) **Thymol turbidity test**

    Although the plasma globulins undergo no great quantitative change in liver disease there are changes in the different globulin fractions which alter their electrophoretic pattern and these are thought to be the basis of the flocculation tests Positive results are found in many cases of active liver disease Similar tests which are not used so frequently are the

Takata Ara colloidal gold and the cephalin cholesterol flocculation tests

(c) **Plasma prothrombin** Since prothrombin is formed in the liver from vitamin K the prothrombin index (p 158) is reduced in obstructive jaundice when vitamin K is not absorbed and in advanced liver disease when prothrombin is not formed in the liver A low prothrombin index persisting after injections of synthetic vitamin K (menaphthone) suggests severe impairment of liver functions

(3) Test depending on excretory functions

Alkaline phosphatases These are enzymes which form inorganic phosphates from phosphoric acid esters It is thought they are formed in bone They are excreted by the liver Their concentration in the blood is increased in bone diseases such as osteitis fibrosa hyperpara thyroidism bone neoplasms and rickets and also in jaundice particularly obstructive jaundice The normal serum concentration is from 1 5 to 4 units in adults and 3–13 units in children

Liver function tests are employed (1) *to assist in the differential diagnosis between medical jaundice (hepatitis) and surgical jaundice (obstructive)* The diagnosis is primarily made upon con sideration of the history and physical signs but complementary tests are valuable Absence of urobilinogen from the urine (p 227) indicates complete obstruction This rarely persists for long in hepatitis and is usually intermittent in gall stones Thus per sistent complete obstruction demonstrated by repeated negative urinary urobilinogen tests favours obstructive jaundice due to growth The Van den Bergh reaction gives no assistance

In jaundice due to extra hepatic obstruction liver function is normal at first and fails gradually In acute hepatitis liver dysfunction is maximum at first and usually lessens Thus in chronic jaundice repeated liver function tests showing deterioration in function suggest obstructive jaundice while tests showing im provement of function suggest hepatitis

The non specific flocculation tests—thymol turbidity etc —are positive early in hepatitis but not in obstructive jaundice whereas the alkaline phosphatase is raised early in obstructive jaundice but

not in hepatitis Thus alkaline phosphatase levels above 40 units with negative or weakly positive flocculation tests indicate obstruction while phosphatase levels below 15 with positive flocculation tests indicate hepatitis

(2) *To detect liver failure either to confirm a clinical diagnosis of hepatic disease or to estimate prognosis when operation is contemplated* For this purpose several tests should be performed In advanced cirrhosis and in hepatic failure from other causes typical findings are a low plasma albumen (less than 4 0 grm per 100 ml) a low prothrombin index raised plasma bilirubin and a raised alkaline phosphatase level

(3) *To detect active disease of the liver* In active disease of the liver i e any form of hepatitis the non specific flocculation tests are usually positive Reliance must not be placed exclusively upon the results of liver function tests Clinical assessment is still the primary diagnostic method

V EXAMINATION OF VOMIT

The character of the vomit varies with the nature of the food ingested and the absence or presence of bile In pyloric stenosis the vomit is apt to be copious sour smelling and exhibits a froth on the surface after standing The presence of much *mucus* gives to the vomit a viscid consistence The appearance of the vomit in hæmatemesis varies If the bleeding is copious the vomit may present the appearance of pure blood and contain clots Such bleeding may come from a gastric ulcer or from the œsophageal varices of portal obstruction More commonly the blood is altered in colour by being in contact with the gastric juice it may be blackish in colour or dark brown The latter appearance is due to the conversion of hæmoglobin into hæmatin The altered blood gives to the vomit an appearance often compared to that of *coffee grounds* The taking of preparations of iron or red wines may produce a similar appearance in the vomit Vomit which contains dark green bile may resemble vomit which contains blood On diluting with water however the green colour of the bile becomes more apparent while blood remains dark If there is any doubt the tests for blood described under the examination of fæces may be applied Remember that blood in vomit may have come from the nose or lungs and have been swallowed Fæcal vomit characteristic of

intestinal obstruction is brownish black in colour and may resemble altered blood but has a typically fæcal odour

VI EXAMINATION OF FÆCES

Examination of the fæces is an investigation of great importance too frequently omitted No patient with bowel disturbance has been properly examined until the stools have been inspected The white surface of a bedpan makes an ideal background for the detection of blood pus and mucus

1 **Naked eye inspection** —The following points should be noted

(a) Amount of the daily stools
(b) Their colour
(c) Their odour
(d) Their consistence and form
(e) The presence of any abnormal constituents

Regarding amount it is sufficient to state whether the stools are copious or scanty The average daily amount of fæces in health is 120–180 grm (about 4 oz)

The **colour** of normal fæces is partly due to stercobilin partly to chlorophyll and other pigments **Black stools** may be produced by the ingestion of iron or bismuth In hæmorrhage occurring high up in the intestine the altered blood makes the stools dark tarry looking and very offensive and the guaiac and benzidine tests are strongly positive

Pallor of the stools may be due to lack of entrance of bile into the intestine as in obstructive jaundice to dilution and rapid passage of the stool through the intestine as in diarrhœa or to an abnormally high fat content as in sprue

The **odour** of the fæces is due to the presence of indol and skatol The absence of bile favours putrefaction hence the stools in jaundice are often very offensive Cholera stools on the other hand contain very little organic matter and are almost free from odour The stools of acute bacillary dysentery are almost odourless while those of amœbic dysentery have a characteristic odour something like that of semen

The **form and consistence** of the stools is of importance In obstinate constipation they may be much drier and harder than

normal and even friable In all forms of diarrhœa they are more fluid than normal and may even be watery Slimy stools are due to the presence of an excess of mucus

Note whether the stools are formed or fluid and if formed if there exists any abnormality in the shape The stools of constipation have often the form of round balls which are frequently coated with mucus In obstruction in the large intestine the stools may be ribbon like The presence of a rectal growth may produce a groove or furrow along the fæcal mass

To detect **abnormal ingredients** the stool should be placed on a fine sieve and a large quantity of water added The whole is shaken and stirred up till the soluble parts are all washed away The residue is then examined **Gall stones** are easily recognized Observe whether they are faceted or not if they are then the stones are multiple Particles of undigested food fruit stones foreign bodies concretions—e g those produced by magnesia—and parasites should all be sought

Full consideration of the parasites which may be found in the stools is undertaken later but it is convenient here to describe the method of searching stools for the **head of a tapeworm** Add to the stool a considerable quantity of water containing a little carbolic acid and shake the mixture gently for a few moments Then allow to stand for about ten minutes The parasite sinks to the bottom the supernatant fluid is poured off and more water added and poured off after standing till the residue is nearly colourless The parasite will then be found The head is about as large as that of a large pin and the neck about as thick as a stout thread

Watery stools are found in all cases of profuse diarrhœa and after the administration of hydragogue cathartics To the stools of cholera the special name of *rice water* stools is applied Such a stool is colourless almost devoid of odour alkaline in reaction and contains a number of small flocculi consisting of shreds of epithelium and particles of mucus The name is applied to it from its resemblance to the water in which rice has been boiled **Purulent** or pus-containing stools are found in severe dysentery or ulcerative colitis or in cases where an abscess has found its way into the intestines **Slimy** stools are due to the presence of an excess of mucus and point to an affection of the large bowel The mucus may envelop the fæcal masses or may be intimately mixed with them **Bloody** stools vary in appearance according to the site of

the hæmorrhage If the bleeding takes place high up the stools look like tar In an intussusception they may look like red-currant jelly If the hæmorrhage is from the large intestine the blood is less intimately mixed with the fæcal matter and may even be of a bright colour In hæmorrhage from the rectum or anus it may merely streak the fæcal masses The stools of bacillary dysentery consist at first of fæcal material mixed with blood and pus later of blood and pus without fæcal material Those of amœbic dysentery characteristically consist of fluid fæcal material mucus and small amounts of blood The stools of sprue are very large pale and putty like and sometimes frothy Those of pancreatic steatorrhœa are said to set like butter in the bedpan

In muco membranous colitis the motions contain casts of the bowel which are mainly composed of mucin The individual casts vary considerably in size being commonly from 1 in to 6 in long but in exceptional cases attaining a length of 1 ft or more These casts when small may on casual observation be mistaken for segments of tapeworm

2 **Chemical examination —Test for occult hæmorrhage** *Benzidin test* (Gregersen) —*This test is the simplest and most suitable for* routine use It is carried out as follows —

Powders are made up containing 0 2 grm of barium peroxide and 0 025 grm of pure benzidin if put up in wax papers they will keep indefinitely Just before the test one powder is dissolved in 5 ml of freshly prepared 50-per-cent acetic acid solution A button of fæces is taken by means of a glass rod from the centre of the stool and smeared on to a clean glass slide A smear on filter paper from a finger stall may equally well be made A few drops of the solution are then run on to the smear A blue or blue-green colour develops within a minute if the test is positive and the reaction is graded according to the depth of the colour and the time it takes to develop as follows —

Positive—a deep blue colour appearing within 15 seconds
Weakly positive—a greenish blue colour appearing within 30 seconds
Negative—no colour appearing within 30 seconds

Food containing large amounts of blood i e black sausage and liver should be excluded for a few days before test Iron-containing drugs and ordinary meat do not affect the results

Fats in fæces —Fat is present in food as neutral fat or triglyceride

It is split to greater or lesser degree by lipases mainly of the pancreas into glycerol and fatty acids Some of the fatty acids if unabsorbed combine with bases to form soaps Fat may therefore be found in the fæces as neutral fat fatty acids and soaps

For methods of estimating fats in the fæces textbooks of biochemistry must be consulted The examination of a single stool is useless If the stools collected over twenty four hours contain more than 25 per cent of dry matter as fat steatorrhœa can be diagnosed but variations in the amount of non fatty residues and *phasic variations in fat absorption make this an uncertain method* In normal circumstances about 20 per cent of the fat in the stools is neutral fat and 80 per cent split fat of which about half is present as fatty acids and half as soaps In steatorrhœa a preponderance of neutral fat has been said to indicate pancreatic steatorrhœa and a preponderance of split fat steatorrhœa due to defects of absorption This is an unreliable method of distinguishing the two types

A fat balance test is the only reliable means of diagnosing steatorrhœa The patient is given a fixed diet of say Ch 250 grm Pr 75 grm F 50 grm and the fat is estimated in the stools collected over several days Normal persons retain more than 90 per cent of the fat and 90 per cent of the nitrogen ingested The distinction between pancreatic steatorrhœa and steatorrhœa due to defects of fat absorption can best be made by combining a nitrogen balance test with the fat balance test Patients with pancreatic steatorrhœa retain less than 90 per cent. of fat *and* nitrogen whereas in those with disorders of fat absorption e g sprue and idiopathic steatorrhœa 90 per cent or more of the nitrogen ingested is retained

3 Microscopical examination —It is best to use a low power objective preferably about 1½ in

Prepare a film as follows Remove a portion of the fæces about the size of a split pea with the end of a match Emulsify it on a slide with a drop or two of saline and apply a cover slip

Muscle fibres are easily recognized by their cross-striation If present in large numbers they indicate defective intestinal digestion

Starch granules are readily detected if a drop of iodine solution is added They are pres nt in excess in the syndrome known as intestinal carbohydrate dyspepsia in which the patient digests starch particularly that of potatoes poorly

Neutral fat occurs as colourless highly refractile droplets or as bile

stained irregular masses which stain with Sudan III and are soluble in ether

Fatty acids occur as sheaves of colourless acicular crystals which dissolve on warming or in ether

Soaps occur as greasy looking amorphous masses or as needles which are shorter and thicker than those of fatty acids They dissolve on warming but not in ether On heating the slide with a drop of acetic acid crystals of fatty acid will be seen to separate out

A simple way of distinguishing fats from mucus or vegetable material is to press down the cover slip If the material be fat the slip remains down if vegetable detritus or mucus it springs back when the pressure is released and air comes in from all around

Normally fat in the fæces is almost entirely in the form of amorphous masses of soap less often as crystals Neutral fat should be practically absent

Mucus occurs as transparent blobs or shreds sometimes bile stained It may contain numerous leucocytes or epithelial cells

For protozoa in fæces *see* p 72

VII INTESTINAL PARASITES

The parasites which occur in the intestinal tract include worms and protozoa Some of the nematode and cestode worms will be described

A NEMATODA

1 Perhaps the commonest of all internal parasites is the thread worm **Oxyuris vermicularis** whose presence is associated with considerable itching about the anus It inhabits the large intestines cæcum and appendix and female specimens can often be seen wriggling about in the recently passed motion of their host To the naked eye they look like small white threads 0 5 to 1 cm in length Under the microscope the female may be distinguished by her much larger size by the large uterus filled with ova and the pointed posterior end whence its name is derived For appearance of ovum *see* Plate 12 15

2 **Ascaris lumbricoides** or round worm has a general resemblance to an earth worm It usually measures from 6 to 8 in but may be up to 13 in long The ova which can occasionally be found in the dejecta have brownish yellow granular contents and in many cases

Plate 12

Intestinal parasites

1 *Entamœba histolytica* Fully developed 4-nucleated cyst containing chromatoid bodies as seen in saline preparations (× 1 500)
2 *Entamœba histolytica* Four nucleated cyst as seen in iodine preparation (× 1 500)
3 *Entamœba histolytica* Active form containing included red blood cells as seen in saline preparations (× 1 500)
4 *Iodamœba butschlii* Cyst as seen in saline preparations Note unstained glycogen vacuole (× 1 500)
5 *Entamœba coli* Fully developed 8 nucleated cyst as seen in saline preparation (× 1 500)
6 *Entamœba coli* Eight nucleated cyst stained by Lugol s iodine solution (× 1 500)
7 *Entamœba coli* Active form as seen in saline preparations (× 1 500)
8 *Iodamœba butschlii* Cyst stained by Lugol s iodine solution (× 1 500)
9 *Giardia lamblia* Cyst form stained by Heidenhain s hæmatoxylin (× 1 500)
10 *Giardia lamblia* Active form stained by Heidenhain s hæmatoxylin (× 1 500)
11 *Trichomonas hominis* Stained by Giemsa s method (× 1 500)
12 *Isospora belli* (*I hominis*) Undeveloped oocyst as passed in human fæces (× 500)
13 *Balantidium coli* Active form stained by Heidenhain s hæmatoxylin (× 350)
14 Ova of *Ankylostoma duodenale* (Hookworm) (× 500)
15 Ova of *Enterobius vermicularis* (Threadworm) (× 500)
16 Ova of *Tænia solium* and *saginata* (Tapeworms) (× 500)
17 Ova of *Trichuris trichura* (Whipworm) (× 650)
18 Ova of *Ascaris lumbricoides* (Roundworm) (× 500)
19 Ova of *Bilharzia hæmatobium* (× 300)
20 Ova of *Bilharzia japonicum* (× 300)
21 Ova of *Bilharzia mansoni* (× 300)

All magnifications approximate

Drawn by M W Cooper

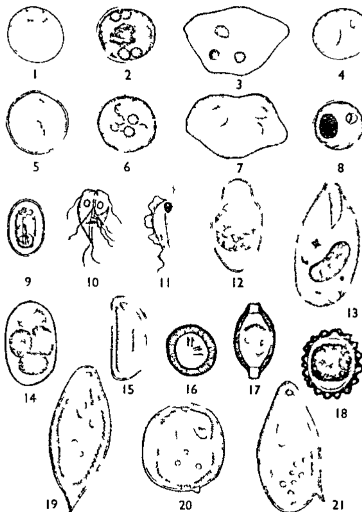


Plate 12 INTESTINAL PARASITES

the shell is surrounded by an irregular albuminous sheath (Plate 12 18)

3 **Ankylostoma duodenale** or hookworm is an important cause of anæmia and debility in the tropics where heavy infestations may occur It is no longer indigenous in Great Britain It lives for the most part in the upper part of the jejunum and its presence there is probable when in an infested district severe anæmia otherwise inexplicable sets in The diagnosis is confirmed by the discovery of ova in the motions (Plate 12 14) They exhibit a segmented yolk enclosed in a thin shell and are sufficiently numerous to be readily detected The adult worm which is but rarely seen before therapeutic agents have been employed is about half an inch long and the mouth is provided with four claw like teeth

4 **Trichina spiralis** gains access to the body as the result of eating infested pork Trichiniasis is rare when pork is eaten cooked but small outbreaks and sporadic cases have been reported in Great Britain and in the U S A When man ingests the muscle trichinellæ of the pig larvæ are set free in the small intestine giving rise to the symptoms of the first stage of the illness—abdominal pain vomiting and diarrhœa The adult female 3 mm long penetrates the intestinal wall and discharges embryos into lymph spaces whence they migrate into muscles In this second stage of the illness the patient has fever and the muscles swell and become hard and tender Death may occur at the height of the myositis Otherwise the embryo undergoes no further development and its capsule becomes calcified Unlike cysticerci described below calcified trichinillæ are not usually visible in X rays

B CESTODA

Many different kinds of tapeworm have been found as parasites in man but the most important are *Tænia saginata* *T solium* and *T echinococcus* Besides its occurrence in the fully developed state *T solium* may be present in the tissues in the form of a cysticercus *T saginata* is never found in this condition in man whilst *T echinococcus* always occurs in the cystic stage and has never been found in the mature condition in the human intestinal tract

The presence of an adult tapeworm in the bowel is generally revealed by the passage of ripe proglottides in the stools and after

the administration of anthelmintics the head may be detected by the methods previously described (p 64)

1 **Tænia saginata (mediocanellata)** is the beef tapeworm Infestation occurs as a result of consuming insufficiently cooked beef infested with the embryo of the worm The adult parasite reaches a length of 4 to 8 metres and consists of about 2 000 segments The ripe proglottides measure 16 by 15 mm The head is quadrate measures 2 mm in diameter has four suckers but is devoid of hooklets The terminal gravid segments of the worm from time to time become separated and the ova are then ingested by the ox in the muscles of which the larva develops It becomes a bladder worm *Cysticercus bovis* measuring 10 by 6 mm and containing an invaginated head which posses es in miniature the characteristics of the adult scolex *Cysticercus bovis* is never found in human muscle tissue or brain For appearance of ovum *see* Plate 12 16

2 **Tænia solium** the pork tapeworm is not encountered in Britain but is endemic wherever infested pork is eaten raw or insufficiently cooked It measures 2 to 4 metres in length a ripe proglottis is 10 by 6 mm The head measures 1 mm in diameter and in addition to four suckers has a rostellum with 32 hooklets The ova in the terminal proglottides are ingested by the pig in the muscles of which the bladder worm *Cysticercus cellulosæ* develops Occasionally soldiers serving in India become infested with *Cysticercus cellulosæ* from eating food contaminated with the ova of the parasite The muscles of the human host are then infested by cysticerci which are palpable through the skin as tense ovoid swellings 10 by 5 mm and of almost cartilaginous hardness About four years after infestation they become calcified and may then be demonstrated radiologically The thigh muscles are those in which cysticerci are most easily demonstrated

3 **Tænia echinococcus** —The adult worm which consists of a head and three segments and whose length is only 3 to 8 mm need not be fully described since it is not found in man The *cystic stage* is very important as it gives rise to serious disease in man in many of the viscera and especially in the liver The cysts of this tænia are not simple but produce from their inner surface one or two generations of secondary vesicles on which the brood-capsules

containing the cestode heads are formed During the period in which this process is going on the primary vesicle dilates to accommodate its increasing contents and may eventually reach the size of a coconut The vesicles may rupture spontaneously and their contents may escape by the lungs by the bowel or by the urinary passages Specimens may be obtained by aspiration or after surgical interference

The diagnosis of suspected hydatid disease may rest upon the recognition of the nature of fluid withdrawn of hooklets or scolices or in the appearance of parts of the ectocyst which are sometimes coughed up from the lungs

The *fluid* is clear alkaline devoid of albumin and contains abundance of sodium chloride and traces of glucose Its density is low being generally under 1010 The *scolex* if it is obtained in a perfect condition is about 1 to 1 5 mm in diameter and a number of them often spring in a group from one brood-capsule They have four suckers and a crown of booklets Portions of the *ectocyst* appear as whitish yellow shreds which can be recognized under the microscope by their lamination and by the pectinate markings on the laminæ

4 **Diphyllobothrium latum (Dibothriocephalus latus)** the fish tape worm is encountered in Sweden Finland and in Michigan The adult worm measures from 3 to 10 metres or more and has a total of 3 000 segments The scolex is small spatula shaped and possesses two deep suctorial grooves The first larval host is a water flea and the second the pike perch or salmon trout Human infestation takes place from eating raw or under-cooked fish

C TREMATODES

Schistosomes or blood flukes are the most important trematode parasites of man They are found in three varieties and produce the disease known as schistosomiasis or bilharziasis *Bilharzia japonicum* and *Bilharzia mansoni* inhabit the portal blood stream and the ova (Plate 12 20 21) are passed in the fæces *Bilharzia hæmatobium* characteristically inhabits the vesical plexus so that the ova (Plate 12 19) are passed in the urine (p 242) They may occasionally also be found in the fæces *Bilharzia mansoni* is found in Africa and South America *Bilharzia hæmatobium* in Africa and the Near East particularly in Egypt and *Bilharzia japonicum* in the Orient

D PROTOZOA

A number of protozoa many of them non pathogenic have been found in the fæces Of these the most important clinically is *Entamœba histolytica* (Plate 12 1 2 and 3) which causes amœbic dysentery (as opposed to bacillary dysentery) and sometimes tropical abscess of the liver *Entamœba coli* (Plate 12 5 6 and 7) is non pathogenic

Entamœba histolytica is found in the stools in two forms In acute attacks vegetative amœbæ can generally be found whilst in the more quiescent stages cysts are passed

If amœbic dysentery is suspected a stool should be passed into a clean bedpan which must be free from antiseptics and the stool must not be mixed with urine It should be taken immediately to the laboratory so that it is examined whilst warm

With a platinum loop select a piece of blood stained mucus or failing this a small particle of fæces emulsify it with a drop of warmed normal saline and apply a cover slip

The diagnosis of vegetative *Entamœba histolytica* depends for practical purposes on the demonstration of actively motile amœbæ which contain red cells The slide must be examined on a heated stage or if this is not available it may be kept warm by applying halfpennies that have been heated in a Bunsen flame on each side of the cover slip Care must be taken not to overheat it Motile amœbæ are readily seen under the low power and can be studied further under the ⅙ objective Iodine will kill the amœbæ and must not be used

Cysts can be seen under the low power as small round refractile bodies They are seen even better if the stool is emulsified in 1 per cent aqueous eosin when provided the stool is fresh they show as white bodies against a pink background The characteristic chromatoid bodies of *E histolytica* are well shown by this method Globules of oil or fat which may be present in patients who have been given oil as an aperient may resemble them and if numerous may make any attempt at further examination useless Oil droplets vary in size are structureless and their edges cannot be sharply focused If cysts are present iodine should be used for their further identification Make a further preparation using 1 per cent Lugol s iodine Find a suspected cyst under the low power Apply the ⅙ objective and centre the cyst in the middle of the field Rack up the microscope tube apply a small drop of oil to the cover slip without moving it and carefully lower the oil immersion objective into the drop The main differences between *E histolytica* and *coli* and their cysts are shown in the table opposite

Besides *E coli* there are three other non pathogenic amœbæ which

	E t mœb hi t lyti Veg tat e f m	*E t mœb c li* V g t u f rm
1 Occurrence	Fairly abundant when present in amœbic dysenteric stools	Never abundant Occasionally seen in dysenteric stools
2 Size	Variable average ₄0 μ to 30 μ	Less variable on the average rather larger than *E h*
3 Motility	Active large pseudopodia These become blunter as the activity diminishes before death	Sluggish Blunt pseudopodia
4 Cytoplasm	Homogeneous and ground glass like (apart from food granules) The differentiation of ectoplasm and endoplasm may be clearly seen Red blood corpuscles often seen	Appearance porcellanous Ectoplasm less plentiful and line of demarcation between it and endoplasm inconspicuous Endoplasm granular abundant food vacuoles with usually bacterial inclusions Red blood corpuscles never present
5 Nucleus	Fainter than in *E c* Karyosome *central* with clear halo round it Periphery marked by ring like layer of regular sized chromatin granules	Distinct Karyosome *nearly always eccentric* halo more definite Ring like layer of peripheral granules more pronounced and irregular in size and shape

	Ent mœb hi t lyti Cy t	*E t mœb c li* Cy t
	When mature four nuclei with nuclear karyosomes central Peripheral chromatin often semilunar Size slightly smaller on average than *E c* cysts Average size 10 μ to 0 μ Glycogen less abundant Refractility moderate Rod shaped chromidial bodies usually seen in fresh specimen Cyst wall rather thinner	When mature eight nuclei with nuclear karyosomes usually eccentri Size slightly larger than *E h* cysts Vary from 10 μ to over 20 μ Glycogen more abundant Refractility considerabl Chromidial bodies not often present thread like or in bundles when seen Cyst wall rather thicker

must not be mistaken for *E histolytica* They are *Endolimax nana* *Iodamœba butschlii* and *Dientamœba fragilis* In the diagnosis of the vegetative forms this mistake will be avoided if it is remembered that *E histolytica* alone is actively motile and contains red cells The cysts of *Iodamœba butschlii* (Plate 12 4 8) contain a single small nucleus a large compact mass of glycogen but no chromatoid bodies

Giardia lamblia is a flagellate protozoon which inhabits the duodenum and may be found in the stools of patients with diarrhœa in both cystic and vegetative form (Plate 12 9 10) There is some doubt whether or not it is pathogenic The fact that a course of mepacrine will eradicate it sometimes with relief of the diarrhœa suggests that it is

Trichomonas hominis (Plate 12 11) is another flagellate protozoon which may be seen in the stools in diarrhœa It is probably non pathogenic Similar if not identical trichomonas may be found in the vagina in leucorrhœa and in the mouth in oral sepsis

Isospora belli (*I hominis*) (Plate 12 12) one of a group of parasites which may produce coccidiosis in man animal and birds has occasionally been described as the cause of acute diarrhœa in man particularly in the Eastern Mediterranean

Balantidium coli (Plate 12 13) is a large ciliate protozoon which is found in the intestine of pigs It occasionally infects man and may rarely cause severe diarrhœa or frank dysentery

CHAPTER IV

THE CIRCULATORY SYSTEM

I ANATOMY

THE heart *lies obliquely in the thorax being inclined from above* downwards forwards and to the left (see plate facing p 44) Two-thirds of it lie to the left of the middle line The part which reaches highest in the thorax is the left auricle It is usually opposite the 2nd interspace or lower border of the 2nd cartilage The greater portion however of the left auricle lies posteriorly and constitutes the hindmost cavity of the heart

The right auricle is the chamber that lies most to the right It extends somewhat beyond the right margin of the sternum and its border may be traced by a curved line joining the 3rd and 7th right sterno-costal articulations and reaching about 1 in to the right of *the sternum*

The right ventricle occupies the great portion of the front of the heart Its inferior margin extends from the 7th right sterno costal articulation to the apex and constitutes the lower border of the heart

The left ventricle only appears in front as a narrow strip scarcely ½ in broad and its outline completes that of the heart on the left where its border forms a curved line ascending from the apex to the lower margin of the 2nd left interspace at a point just internal to the parasternal line The topographical anatomy of the valves of the heart and of the great vessels will be discussed in connection with auscultation as it is in this department that a knowledge of their situation is most necessary (pp 85 88)

The most important organs which come into relation with the heart are the lungs on either side the liver below and the great vessels above A small portion of the anterior surface is only separated from the thoracic wall by the anterior mediastinum whilst behind the heart is in relation with the structures that occupy the posterior mediastinum

That portion of the anterior aspect of the chest which overlies

the heart is known clinically as the **præcordium** It is an area of ill defined extent

It is often necessary to define the exact situation of a point on the front of the thorax and certain landmarks some natural and some artificial are commonly made use of for this purpose

The ribs and interspaces on either side form convenient horizontal landmarks In order to count them feel for the ridge which marks the junction of the manubrium with the body of the sternum known as the angle of Louis or sternal angle When thus has been found run the finger outwards until it reaches the 2nd costal cartilage which articulates with the sternum at this level The space immediately above this is the 1st intercostal space The spaces should then be counted downwards well away from the sternum where they are more easily felt

In order to define the distance of any given point from the mesial sagittal plane of the body a series of vertical lines may be drawn on the chest These are the **midsternal and lateral sternal lines,** down the middle and each border of the sternum , the **midclavicular line** defined as the vertical line dropped from the centre of the clavicle or what amounts to the same thing the line midway between the middle of the suprasternal notch and the tip of the acromion the **anterior mid and posterior axillary lines** descending from the anterior border the centre and the posterior border respectively of the axilla and the **scapular line** which is defined as the vertical line drawn through the inferior angle of the scapula when the arm hangs by the side

The methods commonly employed in the examination of the heart are *inspection palpation percussion* and *auscultation* These will be taken up consecutively although in practice inspection and palpation are often advantageously combined

II INSPECTION

For inspection of the chest the patient should be stripped to the waist set in a good light and examined first standing or sitting up and then lying on his back The observer should directly face him Sometimes it is better to take up a position at the top of the bed and lower one s head and so look along the chest tangentially By this manœuvre various pulsations may be studied with greater ease

The following points must then be noted systematically —

1 The shape of the præcordium
2 Pulsations in the præcordium
3 Bulging or pulsation outside the præcordium either at the root of the neck or the front of the chest or the epigastrium
4 The presence or absence of distended veins on the chest wall or in the neck

1 **The shape of the præcordium** —*In health the chest is bilaterally* symmetrical and there is no greater prominence on the left side than on the corresponding area of the right

Should prominence be observed note whether the ribs are involved or whether the intercostal spaces alone bulge The latter condition occurs rarely in pericarditis with effusion **Prominence of the præcordium** in adults is usually due to deformity of the thoracic cage associated with scoliosis In children it may be produced by great enlargement of the heart or by a pericardial effusion

2 **Pulsations in the præcordium** —Besides the movement of respiration which affects the præcordium with the rest of the chest an impulse which occurs three or four times to each respiration is generally to be seen in the lowest and outermost part of that region This pulsation is called the **apex beat**

The apex beat may be abnormal in **force** in **position** or in **extent** Even in perfect health if the chest wall is thick or if the apex lies behind a rib it may be quite invisible In emphysema the heart is partly separated from the chest wall by the lungs which are increased in volume Absence therefore of the apex beat is not to be regarded as necessarily indicative of disease On the other hand the apex beat may appear to be *more forcible* than usual in cases where the heart is overactive as a result of nervousness or exercise or where the left ventricle is hypertrophied Such changes are more accurately observed by palpation and will be discussed under that head

The **position of the apex beat** may be altered in three classes of cases The cause may be (*a*) *congenital* where the heart is reversed so that the apex lies to the right (dextrocardia) or in those rare congenital heart diseases which give rise to left ventricular enlargement Displacement due to (*b*) *extrinsic causes* where the heart is

displaced by diseased conditions of surrounding viscera which push or pull it from its usual site Instances of pushing are found in pleural effusion and pneumothorax and of pulling in pulmonary fibrosis and collapse of the lung Scoliosis should be remembered as a common cause of outward displacement of the apex beat

Again the displacement of the apex beat may result from (c) *disease of the heart* as in hypertension aortic incompetence and aortic stenosis

It should also be remembered that the position of the apex beat varies considerably with the patient s age and build *in children* it is usually as high as the 4th interspace *in persons with long narrow chests* it may descend as low as the 6th

Pulsation is also seen at times in the 2nd left intercostal space It may ari e either in the pulmonary artery which lies half under cover of the left side of the sternum and half under the inner end of this interspace or it may also be due to aneurysm

3 **Pulsations outside the præcordium** —In addition to the pulsa tions already described movements should be looked for at the root of the neck the front of the chest and the epigastrium

In the suprasternal notch the pulsation is usually systolic in time and when well marked may be an indication of raised aortic arch in hypertension or coarctation of the aorta or of aneurysm of the arch of the aorta

Outside the sternomastoid various pulsations may be observed These may be either arterial or venous The carotids pulsate visibly on exertion from mental excitement in diseases which cause overaction of the heart such as exophthalmic goitre in cases of aortic incompetence hypertension and aneurysm of the aorta In hypertension especially in women and in association with a high aortic arch the right carotid sometimes shows abnormal pulsation due to kinking which must not be mistaken for aneurysm

The jugular veins normally show slight pulsation but if it is excessive and the veins are distended overfilling of the right auricle may be assumed or rarely actual regurgitation through an incompetent tricuspid valve It will be discussed under the head of Venous Pulse (p 106)

Sir Thomas Lewis showed that observation of the cervical veins could be used to estimate directly the hydrostatic venous pressure

and thus form a valuable early sign of congestive heart failure When the external jugular veins are carefully inspected in a normal person the column of blood filling them has a definite upper level This level indicates the position in which the pressure within the vein is equal to that of the atmosphere and its actual perpendicular height above the right auricle is an indication of the right auricular venous pressure In a normal person in the upright position the

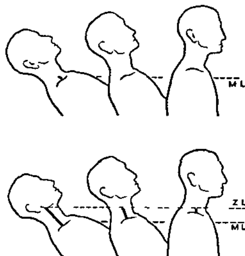


Fig 11

I he ℓth t cal h ght of column f bl d in j g l is bo l l w h m b m terni (M L) wh t th p tion f th p t t In h rt f l wh n th right ic l no pres nc sed th rt l h ght of th l mn (Z.L.) cre sed nd is bo the m b ial l l (M L) wh t th p t f th p tie t (M dif d f m Lewi *Dis as f th H t* 2 d d Lo d)

column is usually invisible With the subject reclining at an angle of 45 the column of blood may extend about one quarter of the distance from the clavicle to the angle of the jaw while with the subject completely recumbent the vein is filled throughout its course in the neck In fact in normal persons the vertical height of the column is about the same as that of the manubrium sterni whatever the position of the subject (Fig 11 M L)

In the presence of venous congestion the hydrostatic pressure is increased so that the vertical height of the column is higher than that of the manubrium In cases of moderate congestion the level of filling of the vein may be seen halfway up the course of the external jugular with the patient recumbent at an angle of 45 (Fig 11 Z L) and in severe cases it may be distended throughout its length with the patient upright Another method of detecting venous congestion is to apply light pressure over the right hypo chondrium In the normal person this produces little change in the neck veins In the presence of venous congestion pressure over an engorged liver produces a rise in the level of the venous column in the neck (the hepato jugular reflux)

Two precautions are necessary in using these methods First, the presence of any mechanical obstruction to the venous return must be excluded Secondly one must demonstrate that the external jugular lies superficially enough to be visible throughout its course in the neck when filled with a column of blood The vein should therefore be compressed with the finger just above the clavicle when it will be seen to fill throughout its length to the angle of the jaw if its course is superficial If however the vein runs deeply in any part of its course it will not be visible and this fact must be allowed for in interpreting the observations on the height of its filling described above Distension of the veins of the neck may also be due to mechanical interference with the venous return to the right auricle as in superior mediastinal obstruction or constrictive pericarditis when pulsation of the veins is also likely to be reduced or absent Distension of one external jugular vein is occasionally seen in health as a congenital anomaly

In the thorax an occasional source of pulsation in unusual parts is *aneurysm of the aorta* Such aneurysmal pulsations always manifest themselves at first above the level of the 4th rib though at a later period they may effect a considerable portion of the chest wall The position of the impulse varies according to the part of the aorta which is diseased If the *ascending aorta* is affected the pulsation is chiefly to the right of the sternum whilst the *transverse aorta* gives rise to less distinct pulsation under the manubrium sterni and the *descending aorta* still more to the left The time of this pulsation is systolic following immediately on the apex beat and it may be observed to be expansile in character

In coarctation of the aorta a collateral arterial circulation develops

and pulsation may be detected in superficial arteries in the chest wall This may best be seen over the back with the patient bending forward to touch his toes

Pulsation in the **epigastrium** is most commonly due to nervousness or excitement in a thin person Less commonly it is due to an hypertrophied right ventricle or to transmission of the aortic pulsation by a tumour such as a carcinoma of the stomach Occasionally it is due to distensile pulsation of the liver in heart failure with tricuspid incompetence and very rarely to an aneurysm of the abdominal aorta which may be palpable as an expansile swelling

4 **Conspicuous veins** —The veins of the thoracic wall may be unduly conspicuous This occurs (*a*) when the patient s skin is unusually transparent (*b*) when an intrathoracic growth or aneurysm obstructs the return of blood to the heart (*c*) when in consequence of portal obstruction or obstruction of the inferior vena caval system the blood returning from the abdominal viscera or lower limbs is forced to find its way through collateral channels

III PALPATION

Before proceeding to the examination of the heart the observer should feel the pulse (p 99) and should confirm the nature of any pulsation seen in the neck Visible pulsation in the neck may be either arterial or venous On palpation *arterial pulsation* is easily felt and produces a thrusting sensation beneath the fingers *Venous pulsation* on the other hand is barely palpable or indeed not felt at all In some cases both types of pulsation may of course be present

In cases of aortic aneurysm the pulsation of the aneurysm may be transmitted to the trachea producing a **tracheal tug** In order to elicit this the observer should stand behind the patient place the tips of his index fingers beneath the cricoid cartilage and lift the larynx and trachea upwards towards the head A distinct tug with every heart beat will be felt if the sign is positive Sometimes this sign can be elicited better in the following manner The patient is asked to swallow and when the cricoid cartilage has moved upwards in the act of deglutition it is caught between the fingers and held in the elevated position By this means the larynx and

trachea are pulled upwards farther than by the previous method and any tracheal tug that is present will easily be felt Care must be taken that the local pulsation of the thyroid arteries is not mistaken for tracheal tug

For palpation of the præcordium the patient should be in an attitude which is easy to maintain since the exertion which a constrained position demands is certain to increase the observer s difficulties When a patient is lying down he should keep on his back because shifts to right or left of the apex beat are easily caused by similar movements of the body

The position of the observer is as important as that of the patient He should stand or sit on the right hand side and then place his right hand which must be thoroughly warm on the patient s chest To begin with the whole palm of the hand should be in contact with the chest wall The finger tips should not be dug unnecessarily into the intercostal spaces as this causes discomfort and may so interfere with subsequent observations When however pulsation is detected over any part of the region under examination its exact position is best determined by the pulp of the fingers

The first pulsation to attract attention is that of the **apex beat** Often the fingers will determine that this is farther from the middle line than inspection would have led one to suppose In such a case the apex beat is taken as the lowest and outermost point at which the finger is distinctly forced up with each beat of the heart The extent and character of the impulse must then be studied It lies in health in the 5th space about half an inch inside the mid clavicular line (p 77) In addition the apex beat may be found to differ from the normal in possessing a forcible or *heaving character* or a *feeble* impulse The pulsation of the apex is sometimes so feeble as to be imperceptible when the patient is lying down but often becomes distinct if he sits up and still more so if he leans forward If however these postures are uncomfortable for a patient who is seriously ill it is better to forgo such advantages than to tire him The chief causes of impalpable apex beat are (*a*) obesity or a thick chest wall (*b*) emphysema of the lungs and less often (*c*) a feeble heart

An unduly *forcible* apex beat may be due to nervousness excitement exertion or thyrotoxicosis but in these cases it does not have the sustained thrust or heave which is felt in left ventricular hypertrophy due to hypertension aortic stenosis or incompetence

Similarly when the right ventricle is hypertrophied as in severe mitral stenosis and in advanced pulmonary heart disease systolic pulsation may be felt in the 2nd and 3rd left intercostal spaces over the infundibulum of the right ventricle and pulmonary artery This phenomenon has been referred to as right ventricular thrust The apex beat in mitral stenosis may be *tapping* in character

In addition to pulsation vibrations may sometimes be felt over the præcordium Such vibrations are termed **thrills** A thrill has been aptly compared to the sensation produced when the hand is placed on the back of a purring cat Single shocks which are really palpable heart sounds do not constitute a thrill The time of occurrence of thrills in relation to the apex beat or to the carotid must be d termined When they commence with the apex beat and continue during the period of ventricular contraction they are termed **systolic** if they are felt whilst the ventricles are relaxed they are termed **diastolic** if they occur near the close of diastole they are termed **presystolic** A diastolic or presystolic thrill felt at the apex indicates mitral stenosis A thrill in the pulmonary area is invariably systolic in time and indicates either pulmonary stenosis or patent ductus arteriosus A thrill in the aortic area is also systolic in time signifying aortic stenosis

IV PERCUSSION

Percussion of the heart as a means of detecting slight changes in the size of that organ or of its chambers has been shown to give false results For these purposes X ray methods are now used (p 127) The student must however learn to percuss the cardiac dullness in order to detect the presence of large pericardial effusions and aortic aneurysms and the absence of the cardiac dullness in hypertrophic emphysema

By percussing in the 4th interspace from the left lung towards the heart it is possible to define the left border more or less precisely It is found about half an inch internal to the midclavicular line The right border of the heart is just to the right of the right lateral sternal line at the level of the fourth rib It is difficult to define since the sternum acts as a sounding board

In aortic aneurysm a dull area can often be mapped out It is continuous below with that of the heart, above it bulges outwards to the right of the sternum at the level of the 2nd interspace and

adjacent ribs whilst the sound produced by percussion of the manubrium terni is also rendered less resonant or even absolutely dull when the aneurysm is large

In *pericarditis with effusion* the dullness varies with the amount of fluid which is present but in well marked cases is pear shaped with the broader end downwards and the upper end higher and broader than the ordinary upper limit of dullness

In hypertrophic emphysema the area of cardiac dullnes is reduced

V AUSCULTATION OF THE HEART AND VESSELS

The stethoscope —Auscultation though sometimes performed with advantage by the direct application of the ear to the chest wall is generally conducted by means of a stethoscope

Stetho copes are of two types single and binaural the former being for most purposes obsolete

In the choice of a binaural stethoscope the ear pieces should receive close attention They should fit comfortably and exclude extraneous sound almost completely Bell and diaphragm chest pieces are available The latter are larger and therefore pick up more sound The diaphragm does not transmit low pitched sounds and hence this chest piece is most helpful in listening for soft high pitched sounds e g the diastolic murmur of aortic incompetence The bell transmits low pitched sounds well e g heart sounds especially the third sound and the diastolic murmurs of mitral valve disease It is convenient to have the bell and diaphragm combined in a single chest piece and selected by a tap Both bell and diaphragm should be large enough to be really useful and the tap should give as direct a connection as possible Thick and short rubber tubes give the least loss of conduction of high pitched sounds

The cardiac cycle and surface anatomy of the valves and vessels — In order to understand the various sounds which can be heard by listening to the heart a clear conception of the events which occur during a cardiac cycle is essential

After the completion of a beat the auricles and ventricles are both relaxed Thereafter the auricles contract forcing blood through the cuspid valves into the ventricles and filling them The ventricles then contract in turn expelling the blood into the vessels whilst

the auricles begin to relax and become refilled with blood finally the ventricles relax also and so the cycle is completed There is thus in rapid succession auricular systole ventricular systole and ventricular diastole auricular diastole commencing during ventricular systole and ventricular diastole continuing through auricular systole

The beginning of ventricular systole is marked by the closure of the mitral and tricuspid valves which had remained open during the systole of the auricles and by the occurrence of the apex beat the beginning of ventricular diastole is marked by the closure of the aortic and pulmonary valves which remain closed until the beginning of the following ventricular systole The carotid pulse occurs a short time after the commencement of ventricular systole in the radial artery it is decidedly later and therefore the radial pulse must never be used to time events in the cardiac cycle The carotid pulse or the apex beat should be used for this purpose

Fig 12 shows the details of the cardiac cycle and their exact timing with relation to an electrocardiographic tracing and to the heart sounds recorded by phonocardiography It will also be found of value when the student arrives at the consideration of the venous pulse (p 106)

In clinical language the words systolic and diastolic are used with reference to the ventricles events which take place during the auricular contraction being described as diastolic (or presystolic) Clinically the systolic phase of the cycle begins with the apex beat and commencement of the first sound It terminates immediately before the second sound whose beginning marks the beginning of the diastolic period

In addition to a knowledge of the cardiac cycle auscultation assumes knowledge of the situation of the valves of the heart and of the course of the principal arteries as well as of the areas where sounds produced at the valve are best heard For full particulars the student must consult works on regional anatomy The following summary recapitulates the important facts —

The pulmonary valve lies horizontally at the level of the upper border of the 3rd left costal cartilage the right half of the valve lies under cover of the sternum the remainder passes outwards behind the costal cartilage (Fig 13)

The aortic valve lies farther from the surface and at a slightly lower level Its situation may be indicated on the front of the chest by a line

drawn obliquely across the left half of the sternum on the level of the lower border of the 3rd costal cartilage

The mitral valve lies slightly obliquely behind the inner end of the 4th left costal cartilage and adjoining part of the sternum The tricuspid

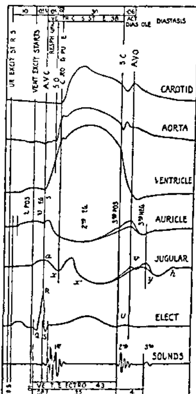


Fig 12 —The cardiac cycle in relation to the pressure in the arteries veins and chambers of the heart and to the electrocardiographic curve

(From Lew *M h l m f th H t B t*)

The times t wh h th citat n wa e beg i n l d t icl are rep se ted by vertic l l n the movem nts of th l lso represented in a imilar w y The scale of bsci sæ is 1 mm = 0 015 sec

A V C. and A V O = closure and pe g of r c lo- e tric l l respecti ly S O and S C = ope g nd lo of sem l na lve re pect ly Other ert cal draw at co enient po ts, a d the ch f time-i t l ma ked bove nd below in seconds.

In the j gul c rve a d r present th icular e rotid d veno s-st si w s x x^1 d y the t o ghs which follow them

In the lectrocardiogr ph c curve P represent a ricula syst l Q R S associated with the initial t g s, d T with th final st ge of tric l sy tol

valve is placed much more obliquely its upper end is opposite the 4th cartilage or interspace and its lower near the lower border of the 5th right sterno-costal articulation It marks the line of junction between the right auricle and right ventricle

The **pulmonary artery** is situated at the inner edge of the 2nd left interspace and behind the adjacent part of the sternum At the lower border of the 2nd cartilage it divides into its branches to the right and left lungs The **ductus arteriosus** passes upwards from the left branch to join the aorta

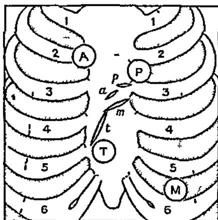


Fig 13 —Position of the cardiac valves and auscultatory areas

The **aorta** arises behind and slightly lower down than the pul monary artery and passing upwards and to the right approaches the surface of the chest most closely at the inner end of the 2nd right costal cartilage arching backwards and to the left from that point

A stethoscope placed over the valves of the heart would fail to distinguish from which a given sound originates because they lie so near each other that the sounds from all of them would reach the chest piece Hence one listens to the mitral valve at the cardiac apex to the tricuspid at the lower end of the sternum to the aortic

over the aorta at the 2nd right costal cartilage and to the pulmonary over the artery in the 2nd left intercostal space It is found that in these regions the sounds of the respective valves are heard most clearly They are therefore called the **mitral tricuspid aortic** and **pulmonary areas** Auscultation should be performed systematically over these areas In ordinary cases begin with the mitral area making certain of the time at which the sounds heard occur in the cardiac cycle by feeling the carotid pulse or the apex beat Then pass to the tricuspid area thereafter to the aortic and lastly to the pulmonary Auscultation may also be performed along a diagonal line joining the mitral and aortic areas This is often useful as for instance when a mitral systolic murmur is associated with an aortic one

In health two sounds are heard over each of these areas the first corresponding with the beginning of ventricular systole the second with the commencement of ventricular diastole The first sound depends on the closure of the mitral and tricuspid valves and on the contraction of the ventricular muscle The second sound is due to the closure of the aortic and pulmonary valves and also but subordinately to tension of the vessel walls This sound is sharper and shorter than the first In health the pulmonary component of the second sound is audible only in the pulmonary area where it can give rise to splitting of the second sound (*see* p 89)

DEVIATIONS FROM THE NORMAL IN DISEASE

In disease the following deviations from the normal may occur —

1 The sounds may have a different intensity both absolutely and relatively to each other from that possessed in health

2 The sounds may be split

3 A triple rhythm may be present

4 Adventitious sounds may be heard either replacing or occurring along with the heart sounds

1 ALTERATIONS IN INTENSITY

In patients with thick chest walls and in those with a serious degree of emphysema the heart sounds may scarcely be audible though there is no heart disease Conversely in the presence of serious heart disease the sounds may appear quite normal Thus alterations in the intensity of the heart sounds are only significant

when considered in relation to all the other features of the case The heart sounds are distant or inaudible in pericardial effusion Accentuation of the first sound is often present in mitral stenosis and may be heard in hypertrophy of the left ventricle and in tachycardia from any cause

Normally in youth the second sound heard in the pulmonary area is louder than that heard in the aortic area In old age the second sound heard in the aortic area is louder (The second sound heard in the pulmonary area is due to closure of both the aortic and pulmonary valves and when closure of these valves is not quite synchronous splitting of the second sound can be heard In health pulmonary valve closure can be heard only in the pulmonary area and is heard most easily in the young) An absolute accentuation of the sound of aortic or pulmonary valve closure may be found in disease when there is an increase in pressure or mass of the column of blood which brings about closure of the valve concerned Thus accentuation of the sound of aortic valve closure is found in systemic hypertension and in aneurysm of the aorta near the aortic valve Accentuation of the sound of pulmonary valve closure is found in conditions which raise the pressure of blood in the pulmonary circulation e g mitral stenosis emphysema progressing to the stage of right heart failure and primary pulmonary hypertension and may give rise to a loud second sound heard in the pulmonary area or splitting of the second sound in which the second (pulmonary) component is louder

2 SPLITTING

Splitting of the first sound is sometimes heard in the mitral area It is often heard in health and is describ d in bundle branch block In itself therefore it is of no pathogenic significance It must be distinguished from triple rhythm to be described below and must not be mistaken as it often is for a short presystolic murmur

Splitting of the second sound is frequently heard in the pulmonary area It results from asynchrony of closure of the aortic and pulmonary valves It is usually audible in children and young persons and the splitting is widest during inspiration and narrowest during expiration Wide splitting of the second sound is often heard in bundle branch block

3 TRIPLE RHYTHM

Electrical recording of heart sounds (phonocardiography) has confirmed that in addition to the two generally recognized a third and also an auricular heart sound are usually present These sounds are usually inconspicuous but can often be detected by careful auscultation When prominent they are responsible for the cadence of sounds known as triple rhythm

The third heart sound follows the aortic component of the second sound by about 0 15 sec It is usually best heard in the mitral area and is lower pitched than the second sound which it follows It is believed to be due to rapid ventricular filling Triple rhythm from a third sound is common in young persons and can occur in health up to the age of 40 It is also heard in heart failure from any cause and sometimes shortly after a cardiac infarct

The auricular sound (previously referred to as the fourth heart sound) occurs before the first heart sound and is associated with auricular systole When it is loud there is often a prominent venous a wave in the neck and a large P wave in the electro cardiogram The auricular sound is not heard in health by ordinary *auscultation Triple rhythm from an auricular sound occurs in* some cases of systemic hypertension and pulmonary heart disease It is not a sign of heart failure and is never heard in auricular fibrillation

4 ADVENTITIOUS SOUNDS

Adventitious sounds may be of two kinds endocardial or exocardial Abnormal endocardial sound are called murmurs Organic murmurs are due to disease either of or close to the valve where they occur Murmurs unassociated with any structural disease in the heart are referred to as functional They may be due to alteration in the viscosity of the blood or to other causes *They include hæmic and cardio respiratory murmurs* An organic murmur may result either from obstruction to the onward flow of the blood or from leakage backwards through a closed but incompetent valve The former are known as obstructive murmurs the latter as regurgitant In examining a murmur the following points must be noted —

(*a*) Its time of occurrence
(*b*) Its point of maximum intensity

(c) Its direction of selective propagation beyond the præcordial area
(d) Its character

The characteristics of some common murmurs are shown diagrammatically in Fig 14

The **time of its occurrence** is noted with reference to the sounds of the heart and these by comparison with the time of occurrence of the apex beat

The *maximum loudness of a murmur which has been produced at* a given valve usually occurs at the point where the valve sound would be best heard in health To this rule however there are some exceptions

Valvular murmurs are not equally well heard at all points of the chest wall which are equidistant from the point of their greatest intensity but each is much more distinctly audible at a distance in some directions than in others i e such murmurs have **directions of selective propagation**

The **character** of the murmur also helps to decide a doubtful case Obstructive murmurs are usually rough regurgitant murmurs softer and blowing The duration of a murmur may also be important For example some murmurs seem to fill systole and may even mask the first and second sounds while others seem to be separated by silent intervals from these sounds The loudness of a murmur has no relation to its importance A very loud murmur may be less serious than one so soft as to be nearly inaudible

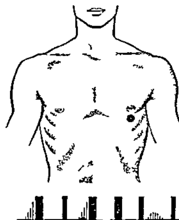
Murmurs due to disease of postnatal origin proceed from the valves of the left side of the heart more often than from those of the right and in adult life murmurs at the pulmonary area due to disease of this valve are rare A description of the more important murmurs and exocardial sounds follows

(1) *Mitral Murmurs*

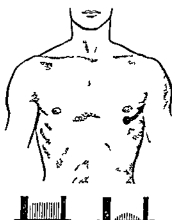
Mitral murmurs may be either (a) diastolic or (b) systolic (Fig 15)

(a) **Diastolic murmurs** are for practical purposes always organic

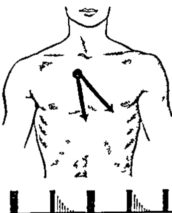
They are of two types The mid-diastolic murmur is separated from the second sound by a brief interval The presystolic murmur begins with auricular contraction Mitral diastolic murmurs are due to the onward rush of blood through the deformed or narrowed [illegible]



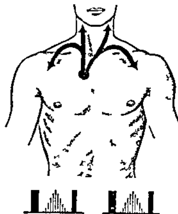
1 M tral presyst lic and mitral mid dia tol c m rm rs (mitral stenosi)



.. M tral systolic murm (m tral incompetence) a d functional syst lic mor m Th first may be ducted to the axilla and ba k Th second is not conducted.



3 Aortic di tolic murm (rtic incompet nce) Th s murm r s som times h ard be t down th left d of the ternum



4 Aortic syst lic murm (rtic ste os) This m rm is occa ionally h d be t th pex.

Fig 14 —To show the usual point of maximum intensity direction of selective propagation and timing of some common murmurs

into the wider cavity of the left ventricle At the beginning of diastole (i e immediately after the second sound) the pressure in the left ventricle is approximately equal to the diastolic blood pressure As the ventricle relaxes the pressure within falls almost to zero until it is less than the pressure in the left auricle and the mitral valve opens Often the opening of a damaged and thickened mitral valve produces an audible sound the opening snap Obviously there must be an appreciable pause between the second sound and the opening snap to enable the pressure changes in the left ventricle to take place Opening of the m tral valve is followed by a murmur due to passive filling of the left vnetricle through

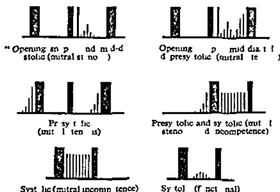


Fig 15 —Mitral murmurs

the narrowed mitral valve The flow of blood diminishes towards the end of diastole and the murmur is therefore mid-diastolic in time Shortly before the first sound the auricle contracts and reinforces the flow through the mitral valve giving a presystolic murmur

Obviously the presystolic murmur will be absent when there is no co-ordinated auricular contraction as in auricular fibrillation Similarly a third sound which is due to rapid filling of the ventricles (and especially the left ventricle) will seldom be heard in pure mitral stenosis

Mitral diastolic murmurs are usually best heard at the apex or just medial to it and are often sharply localized They have no direction of selective propagation They are harsh low pitched and rumbling and sometimes associated with a thrill The first sound in mitral stenosis is usually loud and abrupt

An opening snap is usually present with mitral diastolic murmurs It is a high pitched sound occurring about 0 1 sec after the second sound and best heard at the lower end of the sternum It may be confused with the second component of a split second sound but can be distinguished because it is in fixed relation to the aortic component of the second sound (i e the second sound heard in all areas except the pulmonary) throughout the respiratory cycle Again the opening snap is usually best heard at the lower end of the sternum while splitting of the second sound is confined to the pulmonary area A soft murmur in early or mid systole is often heard at the apex in association with mitral diastolic murmurs Such a murmur does not indicate mitral incompetence

The recognition of the murmurs of mitral stenosis is of great importance and it is especially necessary that splitting of the first sound which is often present in health should not be mistaken for a presystolic murmur In cases of doubt the patient should be exercised until he has a moderate tachycardia and should then lie on his left side Under these circumstances the presystolic murmur of mitral stenosis will be rendered more prominent while splitting will be unaltered or will disappear This manœuvre should be employed in every case in which the diagnosis of mitral stenosis is in doubt

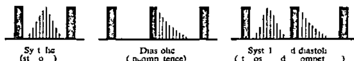
(*b*) **Systolic murmurs** in the mitral area may be due to mitral incompetence and regurgitation but are usually due to other causes The systolic murmur of mitral incompetence begins with the apex beat and may replace more or less completely the first sound in the mitral area The phonocardiogram shows that the murmur fills systole and may increase in intensity in late systole so that clinically it may obscure the second sound Its point of maximum intensity is at the apex and its direction of selective propagation is outward towards the axilla and the angle of the left scapula The murmur is relatively harsh and may rarely be accompanied by a thrill

Other systolic murmurs heard in the mitral area for example functional murmurs and those due to hypertension and anæmia can be distinguished because they are more or less localized to the mitral area and do not have the direction of selective propagation of the murmur of mitral incompetence they are never loud and never accompanied by a thrill they never seem to fill systole and

are usually confined to mid systole Further any murmur which disappears with a change in the patient s position or which dis appears if the breath is held in inspiration can usually be discounted as of no pathological significance

(2) *Aortic Murmurs* (Fig 16)

(*a*) **Systolic murmurs** are due either to stenosis of the aortic valves or to deformity of the valve cusps They are rough in character The phonocardiogram shows that they are loudest in mid systole starting well after the first sound and finishing well before the second sound This gives a typical grunting character to the murmur Aortic systolic murmurs have their area of greatest intensity at the 2nd right costal cartilage near the sternum but may be almost as well heard in the mitral area They are propagated with the bloodstream into the arteries and may in most instances



Sy t lic (st o) Dias olic (incomp tence) Syst l d diastoli (t os d ompet)

Fig 16 —Aortic murmurs

be readily heard over the carotids Aortic st nosis can be diagnosed for certain when an aortic systolic murmur is accompanied by a thrill felt at the aortic area Often a thrill can be brought out in the aortic area by having the patient lean forward holding his breath in full expiration The character of the pulse (plateau pulse) and the position and character of the apex beat are also helpful in making the diagnosis of aortic stenosis

(*b*) **Diastolic murmurs** may replace in part or completely the normal second sound in the affected region They are always organic and indicate aortic incompetence and regurgitation They may be heard in the aortic area not infrequently however they are better heard over the left half of the sternum at the level of the 3rd rib and interspace and lower Their direction of selective propagation is towards the lower end of the sternum though occasionally they are almost equally well heard near the apex (Fig 14) their char acter is soft and high pitched with an echoing diminuendo quality

When there is any doubt about the presence of this murmur the patient should lean forward and hold his breath with the chest in the position of full expiration The observer should then listen with the diaphragm again Sometimes this murmur is heard best if the ear is applied directly to the chest wall

In many instances one finds that a **double murmur** is present at the aortic orifice the systolic element of which is not caused by real stenosis of the ostium but by roughening and deformation of the valve segments the diastolic murmur being due to the backward leakage through the misshapen cusps In these circumstances aortic stenosis can only be diagnosed in addition to incompetence in the presence of a systolic thrill in the aortic area or of characteristic change in the radial pulse

A mitral diastolic murmur indistinguishable from that due to mitral stenosis may occasionally be heard in aortic incompetence in the absence of any disease of the mitral valve This is known as the Austin Flint murmur but is a great rarity

(3) *Tricuspid Murmurs*

These are rare

(*a*) **Diastolic murmurs** have their maximum intensity at the lower end of the sternum They have no selective propagation They are similar in character and timing to mitral diastolic murmurs but increase markedly on inspiration

(*b*) **Systolic murmurs** have a similar character to the systolic murmur of mitral regurgitation They are best heard in the tricuspid area and are due to tricuspid regurgitation They are markedly increased on inspiration which draws more blood into the right heart They are associated with considerable systolic venous pulsation in the neck and with expansile pulsation of the liver

(4) *Pulmonary murmurs*

These are best heard in the pulmonary area The systolic murmurs do not fill systole but are longer than aortic systolic murmurs Organic systolic murmurs are uncommon and may be accompanied by a thrill and often by a prominent a wave in the neck.

Functional systolic murmurs are common and are due to

increased flow in the pulmonary artery in anæmia hyperthyroidism or atrial septal defect or to chest deformity

Pulmonary diastolic murmurs are rare A diastolic murmur heard occasionally in the pulmonary area in mitral stenosis is known as the Graham Steel murmur

(5) *Congenital Murmurs*

(*a*) In a child with congenital heart disease a harsh systolic murmur (often with thrill) in the 2nd left interspace with faint or absent pulmonary second sound indicates **congenital pulmonary stenosis** A combination of pulmonary stenosis ventricular septal defect enlarged right ventricle and dextroposition of the aorta is known as the **tetralogy of Fallot** It is probably the commonest form of congenital heart disease with great cyanosis seen after early life

(*b*) In a child or adult without cyanosis and even without symptoms a peculiar loud continuous murmur (systolic diastolic) in the 2nd left interspace often transmitted towards the neck is characteristic of a **patent ductus arteriosus** A thrill is often associ ated and the pulmonary second sound accentuated

(*c*) A loud blowing systolic murmur heard best at the 3rd left interspace and accompanied by a systolic thrill often indicates a ventricular septal defect

(*d*) A harsh systolic murmur at the base of the heart with enlarge ment of the ascending aorta large arterial pulsation in the upper limb and the opposite in the lower and a collateral circulation may be produced by **coarctation of the aorta**

A patent foramen ovale like certain other developmental defects need not give rise to a murmur

The history cyanosis when present site of the murmur nearer the base than the apex and its usually harsh character in general distinguish congenital from acquired lesions Radioscopy and electrocardiography will help to make a diagnosis

(6) *Cardio Respiratory Murmurs*

Cardio-respiratory murmurs occur in cardiac systole They generally begin about the middle or near the end of that period They are short in duration and are best heard during inspiration but a very full inspira tion may render them faint or inaudible

Their commonest situation is just outside the apex beat In a smaller

number of cases they are only heard at the base of the heart at or near the 2nd left intercostal space Changes in the posture of the patient often cause them to disappear entirely They are of no pathological significance

(7) *Hæmic and Vascular Murmurs*

In anæmia hæmic murmurs frequently occur over the heart and vessels They are usually heard in the 2nd left intercostal space over or just external to the pulmonary area but they are also heard at times in the mitral and much less frequently in the tricuspid and aortic areas the last being uncommon *In all cases such murmurs are systolic in time and usually become less distinct or even inaudible when the patient assumes an upright posture*

Sounds and murmurs are occasionally heard over peripheral arteries Thus aortic systolic murmurs are often conducted up the arteries of the neck A murmur may be heard over the thyroid gland in thyrotoxicosis Murmurs may be heard over aneurysms and arterio venous aneurysms in any situation may be accompanied by continuous murmurs of a humming quality usually louder during systole In children a continuous venous hum may occasionally be heard in the neck and may be conducted down into the upper chest It is abolished by slight change of posture In free aortic regurgitation a systolic sound may often be heard over large arteries It is usually best heard over the femorals and has been referred to as the pistol shot sound

When there is an aneurysmal dilatation of the aorta murmurs are usually absent, except in the presence of aortic incompetence when an aortic diastolic murmur is heard In aneurysm of the ascending aorta a systolic murmur and thrill may occasionally be present as may also a diastolic tap or shock

(8) *Exocardial Sounds*

Exocardial sounds may be due either to pericardial friction or to a localized pleurisy near the heart

When pericardial friction occurs over an area uncovered by lung it has a remarkably superficial character and thus can often be readily recognized

Unlike the murmurs already described pericardial friction does not correspond definitely with the events of the cardiac cycle It is generally more distinct in systole than in diastole but tends to

exhibit a to and fro character the first element occurring during systole and the second during diastole but not necessarily commencing at the beginning of either phase Sometimes the sound occupies the latter part of systole and the early part of diastole without any pause between its first and second elements sometimes it remains audible during the whole of the cardiac cycle Further its position of greatest intensity does not correspond with any of the areas in which valvular murmurs are best heard and it is not propagated but remains confined within narrow limits Its position may vary from day to day The intensity is often considerably modified by the posture of the patient and by the pressure with which the stethoscope is applied

When sufficient fluid is present the friction sound disappears and the cardiac sounds become faint and distant

To distinguish between the rub of pericarditis and that of pleurisy over a neighbouring portion of lung the patient should hold his breath Pericardial friction is unchanged by this but if the friction is of pleural origin it will either be much reduced in intensity or will cease On the other hand deep respiration will increase the pleural sound but will not influence the pericardial Pleuritic and pericardial friction may occur together

VI THE PULSE

The pulse gives direct information regarding the condition of the vessel walls and the amount and variations of pressure of the contained blood By these observations valuable information regarding the state of the heart and circulation as well as of the general state of the patient can be obtained

The patient should not have been making any effort for some little time beforehand The pulse is best felt when the patient s forearm is pronated and the wrist slightly flexed In cases of aortic regurgitation the peculiar character of the pulse is more distinctly brought out when the patient s arm is elevated

The following observations should be systematically made —

1 Rate of pulse
2 Rhythm
3 Character
4 Volume
5 Condition of vessel wall

Estimates of the tension of the pulse that is of the blood pressure within the vessel by palpation are quite unreliable The blood pressure should be determined with the sphygmomanometer

1 The rate of the pulse is stated as so many beats a minute *It is counted not when the fingers are first laid upon the pulse but* when any quickening due to nervousness of the patient has subsided and the pulse has resumed its normal rate Count the beats for not less than half a minute In cases of auricular fibrillation the pulse rate counted at the wrist may not indicate the true rate of ventricular contractions In all such cases the rate of the heart beat should be counted by auscultation at the apex and the difference between this rate and the pulse rate at the wrist should be recorded This difference is referred to as the pulse deficit

2 Decide next whether the **rhythm** is regular or irregular If it is irregular decide if it is completely irregular whether the irregularity has a recurring pattern or whether an otherwise regular rhythm is occasionally interrupted by some slight irregularity Again if it is irregular decide whether the beats are unequal in volume as well as irregular in time The significance of these observations will be clearer when graphic methods of recording the pulse have been considered

3 Study the **character or form** of the individual pulse wave Variations will be described under pulse tracings but some particularly the collapsing pulse of aortic incompetence in which there is a sudden rise in pressure followed by an equally sudden fall can readily be recognized by palpation

4 Estimate the **volume** of the pulse beat that is the amplitude of movement of the vessel wall during the passage of the pulse wave A pulse of large volume is found after exertion and in acute fevers and a pulse of small volume after hæmorrhage

5 **The condition of the vessel wall** For this purpose sufficient pressure should be exerted to empty the vessel of blood and it should then be rolled beneath the fingers against the underlying bone In young persons the arteries cannot be felt or are soft In older persons they are more easily palpable In arteriosclerosis they may feel hard like whipcord and may be tortuous It is a good practice to feel both radials and both brachials

The typical pulse of a healthy adult man should be described in such terms as the following The rate is 70 per minute The beats are regular in rhythm and equal in volume The pulse is of

moderate volume and is not collapsing in character the arterial wall is just palpable but is neither thickened nor tortuous

The **blood pressure** is taken with a **sphygmomanometer** The patient should be sitting or lying at ease The manometer is placed so as to be at the same level as the observer s eye Fix the empty armlet closely to the upper arm which should be thoroughly relaxed The estimation may now be made by one of two methods or by one and then the other

(i) **Palpatory method** for systolic pressure Steadily inflate the armlet until the pulse is no longer felt at the wrist By means of the valve allow the air to escape very gradually so that the pressure falls watching the manometer but concentrating attention upon the return of the pulse at the wrist Immediately the pulse returns read the scale this reading is the *systolic* blood pressure in millimetres of mercury

(ii) **Auditory method** for systolic and diastolic pressures Raise the pressure by pumping to 30 mm or so above the systolic blood pressure as determined by the palpation method If the auditory method is used exclusively the mercury should be blown up to the top of the sphygmomanometer tube Place the chest piece of a binaural stethoscope on the arm immediately below the armlet and auscultate the brachial artery Open the valve and so reduce the compression gradually until the faint tapping sounds produced by successive pulse waves are first heard and immediately take a reading This is the *systolic* blood pressure Continue to listen while the mercury falls the sounds persist and become gradually louder until quite suddenly they become soft and perhaps inaudible Take a reading at this point also it is the *diastolic* pressure

The figures for systolic pressure as obtained by the palpatory and the auditory method are much the same by the latter method the reading is usually a trifle higher (5–10 mm Hg) The auditory method is recommended for general use

Several types of sphygmomanometer are obtainable with an aneroid manometer bearing a dial in place of a column of mercury These are compact and convenient but the mechanism is liable to give false readings especially with the lapse of time It is therefore important to connect it with a mercurial manometer occasionally to make sure that it is accurate

Precautions —Arterial pressure shows temporary variations with

change of posture after meals on exertion and notably a rise on excitement Hence it should be observed only after the patient has been reassured and when he is quietly resting free from excitement and with the arm relaxed In nervous patients the first reading is often too high and should be rejected a second reading will more closely represent the true pressure The pulse rate at the time should be noted for blood pressure varies to some extent with the rate of the heart It is essential to work as quickly as is compatible with accuracy for compression of a limb itself induces a rise in blood pressure To reduce this source of error when successive estimations are to be made the air pressure in the armlet should always be allowed to fall to zero as soon as each reading has been taken

Occasionally the sounds disappear at a point below 200 mm for a period and then reappear finally disappearing at the point of diastolic pressure Thus the sounds may first appear when the mercury falls to 210 (systolic pressure) disappear from 180 to 160 (silent gap) reappear and finally disappear at 120 (diastolic pressure) This phenomenon of a silent gap is found in certain patients with hypertension its significance is unknown but its occurrence makes it important that the armlet pressure should always be well raised at the beginning of an estimation of blood pressure

It sometimes happens that the sounds become indistinct or inaudible in the course of the examination In this event one should remove the cuff and replace it on the opposite arm or on the same arm after a short pause If there is gross œdema of the arm or if the muscles are held contracted the readings are so inaccurate as to be valueless

Occasionally it becomes necessary to compare the systolic blood pressure in the arm with that in the leg The patient lies face downwards and the cuff is applied above the knee and auscultation carried out over the popliteal artery In health it is found that the systolic pressure in arm and leg is approximately the same provided that the subject is horizontal Under the same conditions patients with aortic incompetence may show a systolic pressure in the leg higher than that in the arm while in coarctation of the aorta the systolic blood pressure is low in the leg

Normal blood pressure—The average systolic pressure in healthy adults is 100–140 mm Hg the average diastolic pressure 60–90 mm

In children it approximates to the lower figure in each case and in the elderly it reaches or even exceeds the higher figure The difference between the systolic and the diastolic pressures—the pulse pressure—is 30–60 mm.

Abnormal blood pressure—An abnormally high pressure (*hyper tension*) is found in chronic nephritis and in a large group of patients in whom hypertension may be the leading sign (*essential hyper tension*) Too great importance need not be attached to a single reading which does not greatly exceed the normal limits On the other hand a high systolic pressure such as 200 especially if persistent and accompanied by a high diastolic pressure such as 130 is always significant. A diastolic pressure persistently of or above 100 is considered abnormal for life insurance purposes

An abnormally low pressure (*hypotension*) is not nearly so commonly encountered It may be found temporarily as in hæmorrhage shock, or peripheral circulatory failure from any cause or persistently as in Addison s disease It must be remembered that a low pressure is natural to some persons and is not necessarily a sign of disease

VII EXERCISE TOLERANCE TEST

The best test of the efficiency of the heart is the effect of exercise In its simplest and safest form the test is applied by the patient s walking briskly up a flight of 40 steps or he may hop 20 times on each foot, raising the shoulders 6 in. at each hop The test may also be carried out by his stepping on and off an 18-in stool twenty times

As a result of such a test, there should if the heart is healthy be little disturbance or respiration and the pulse-beat should not rise by more than 10 to 20 beats per minute and should reach the normal again after about one minute It is however important to note that patients with effort syndrome or functional symptoms *referred to the heart, often show more distress in performing this* test than do those with organic heart disease

VIII CARDIOGRAPHIC METHODS

The precise measuring and recording techniques of physics have been applied very successfully to cardiology Special instruments

have brought new advances The most important of the early instruments was the polygraph though it is seldom used nowadays The cardiographic methods in routine used at the present time are electrocardiography and cardioscopy Other cardiographic methods sometimes used for research and the investigation of special problems such as congenital heart disease include cardiac catheterization and angiocardiography

A THE POLYGRAPH

The polygraph consists of two or more tambours recording on paper which is moved by a clockwork mechanism A timing trace recording fifths of a second is also provided Usually the venous pulse in the neck (Phlebogram) and the radial pulse (Sphygmogram) are recorded simultaneously

The sphygmogram —In a pulse tracing rise of blood pressure will be represented by an upstroke and fall by a downstroke The pressure rises fairly rapidly therefore the upstroke when the paper is driven forward at the usual speed is nearly but not quite perpendicular The percussion wave is quickly followed by what is known as the tidal wave these are not separately distinguishable by the finger in health and the



Fig 17 —Normal sphygmogram

perc ssio wa s (partly in trume tal) *t* t dal ma n p lse wave ort c notch *d* dicr tic wa e E, the period of ntric la ystole (ortic al s open)

second wave (the tidal) represents the natural summit rather than the first (the percussion wave) which is largely due to instrumental fling Thereafter the pressure begins to fall off but at the moment when the aortic valves close the decrease of pressure is arrested and a positive (dicrotic) wave is propagated into the vessels this event is recorded as a small break in the downstroke of the tracing The foot of the notch immediately *before* the dicrotic upstroke or wave indicates the point of time when the aortic valves close (aortic notch) After the dicrotic wave the line again curves downwards until a new upstroke

marks the arrival of the next pulse wave Ordinarily the blood pressure requires much longer to fall than to rise hence the downstroke is much less vertical than the upstroke

Certain conditions often present characteristic tracings and the same features are recognizable on feeling the pulse Thus although the polygraph is seldom used at the present time careful examination of the radial pulse may confirm or suggest a diagnosis

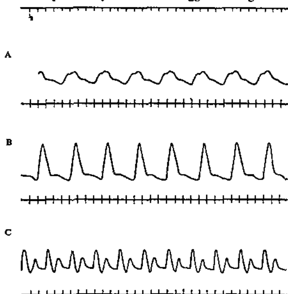


Fig 18 —Sphygmograms showing typical form of pulse wave in (A) aortic s enosis (B) aortic incompetence and (C) pulsus alternans

(*a*) Aortic stenosis —Small amplitude sloping upstroke (plateau pulse) tidal wave well developed and often higher than the percussion wave (anacrotic pulse) (Fig 18 A)

(*b*) Aortic incompetence —Great amplitude abrupt upstroke rapid fall little or no dicrotic wave This is known as the water hammer collapsing or Corrigan pulse (Fig 18 B)

(*c*) Mitral stenosis —Small amplitude if the lesion is advanced Complete irregularity (auricular fibrillation) is often present

(*d*) **Aneurysm** of the arch of the aorta occasionally leads to a diminution in amplitude in the pulse on one side usually the right according to the part of the aorta implicated Other signs of aneurysm would be present and it must be remembered that in health there may be a natural difference between the pulse on one side and on the other

(*e*) **Pulsus paradoxus**—The pulse becomes smaller or even disappears at the end of inspiration when the patient breathes deeply This sign is found in pericardial effusion and in constrictive pericarditis

(*f*) **Pulsus alternans** (Fig 18 c)—When the ventricle beats strongly then weakly in successive beats of normal rhythm alternation is present In the radial tracing are seen alternate large and small beats which are however *equidistant* The polygram and the electrocardiogram are of normal form The condition is often discovered when the systolic blood pressure is taken and the rate of sounds suddenly doubles as the pressure in the cuff falls

When this condition is discovered provided the heart rate is moderate and no abnormal rhythm is present it may be *inferred that the heart muscle is severely damaged*

THE VENOUS PULSE (PHLEBOGRAM)

Pulsation in the jugular vein is physiological and a jugular tracing is obtainable from most people in health as well as in disease Visibility of the pulsation in health varies in different individuals it may not be seen at all when the neck is fat or when the person is standing

Venous pulsation at the root of the neck is best observed when the patient is lying down with the head only slightly if at all raised on a pillow In some cases as in aortic incompetence the pulsation in the neck seems exclusively arterial (carotid) Given the necessary conditions of which the chief is an interference with return of the blood to the right auricle the jugular veins become distended and prominent The venous pulsation then becomes obvious and occasionally it is practicable to make direct observations on its character In complete heart block for instance waves may be seen while the ventricle is in diastole from which it may be inferred that they are auricular waves dissociated from the far less frequent ventricular waves Where functional tricuspid

incompetence occurs in advanced stages of heart disease the distended jugular vein may show pulsation of great amplitude

If the venous tracing obtained from a polygraph (Fig 19) be analysed it will be seen that there are three main waves *a* *c* and *v* The first wave *a* is due to auricular systole in part directly from an impulse sent back to the vein and in part from swelling of the vein whil the contracting auricle holds back the blood in the neck The second wave *c* so-called the carotid pulse is due to closure of the auricular valves and therefore indicates the beginning of ventricular systole Immediately after the carotid wave there is a fall in pressure due to the descent of the auriculo ventricular septum (mid ventricular contraction) which is

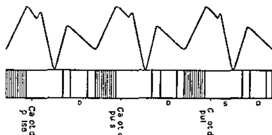


Fig 19 —Diagram of venous pulse

AS l y t l VS t l y t le D d t I (t l)

soon followed by the third wave *v* The *v* or venous stasis wave is due to filling of the auricle which is still closed below by the auriculo-ventricular valves Immediately ventricular systole ends and the ventricle relaxes the blood from the auricle rushes into it hence the *v* wave falls and a final depression in the curve is seen Waves *c* and *v* are together spoken of as the ventricular complex

Normally *a* precedes *c* by not more than [1] sec for this represents the time taken for an impulse from the auricle to reach and affect the ventricle (*a* *c* interval)

The commonest alterations in the venous pulse as the result of disease are—

(i) Absence of the auricular wave *a*—characteristic of auricular fibrillation and associated with complete irregularity of the *c* wave and of the corresponding radial pulse

(ii) Prolongation of the *a–c* interval i e heart block If the block is more severe *a* waves will occur without a consecutive carotid wave *c* (dropped beat) if the block is complete *a* and *c* waves are dissociated entirely

B THE ELECTROCARDIOGRAPH

The action of the excitable tissues of the body is associated with electrical activity Changes in electrical potential associated with the contraction of the heart can be recorded from the body surface but as they are small (about 1 millivolt) special equipment is required

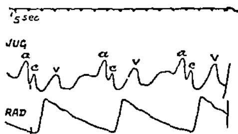


Fig 20 —Normal polygram showing three heart-cycles

a ur c l systol *c* carot d w th beg nni g of nt c l systole venou ta wave at th end of tr c l r tra t n The r d al be t oc rs se aft r th rotid w e

The basis of this equipment is the string galvanometer This consists of a quartz fibre coated with a conducting material which is suspended between the poles of a strong magnet Electric currents derived from the heart flow through the fibre and the powerful magnetic field causes it to move A light source and lens system magnify these movements about a thousand times and they are recorded on photographic film Valve amplifiers can be used to magnify the small currents so that they will operate smaller and more robust galvanometers Some of these record on photographic film and others use a heated stylus recording on specially treated paper so that a permanent record is available at once—the direct writer All instruments provide timing marks on the record at 0 1 or 0 2 second intervals

There must be two points of contact with the body to lead the

electrical activity of the heart to the galvanometer These connections are termed electrocardiographic leads The leads in common use are—

The limb leads (standard or bipolar limb leads)
Lead I right arm—left arm
Lead II right arm—left leg
Lead III left arm—left leg

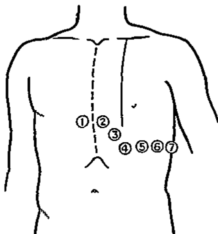


Fig 21 —The position of the exploring electrode for chest leads

The bipolar chest leads use an exploring chest electrode (C) and an indifferent electrode on the right arm (R) or the left leg (F for foot) The various positions of the exploring chest electrode are indicated in Fig 21 Often only three positions are used e g CR1 CR4 and CR7 The indifferent electrode in the CR leads is placed on the right arm Lead CR1 uses a chest electrode at the junction between the 4th intercostal space and the right sternal border CR4 the 5th intercostal space at its intersection with the left midclavicular line CR7 the left posterior axillary line at the same level as CR4

The V (unipolar) chest leads —In the CR leads the electrocardiogram records the voltage difference between the chest electrode and the right arm electrode The latter is not truly indifferent and the CR leads

do not therefore give an exact record of the potentials at the chest wall If such a record is required advantage is taken of the fact that a connection between the right arm the left arm and the left leg is electrically neutral and a truly indifferent electrode The exploring chest electrode is placed as in the corresponding CR lead, the other terminal is connected to a wire joining the limb electrodes either directly or through resistances of 5 000 ohms Such leads are termed V (voltage) leads

The V (unipolar) limb leads use the same indifferent electrode as the unipolar chest leads the other terminal being connected to the right arm (VR) the left arm (VL) and the left leg (VF) in turn

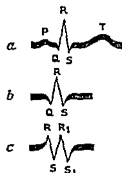


Fig 22 —Terminology of the electrocardiogram

The deflections or waves of the electro cardiogram are designated by the letters PQRST as shown (Fig 22*a*) The P wave is associated with auricular contraction the QRS with ventricular contraction and the T wave with ventricular recovery The Q wave is an initial downward deflection in the QRS complex (Fig 22*b*) If a downward deflection follows an initial upward deflection (R) it is called an S wave and a further upward deflec tion an R^1 (Fig 22*c*)

THE ELECTROCARDIOGRAM IN DISORDERS OF CARDIAC RHYTHM

By providing together a record of auricular excitation (P waves) and ventricular excitation (QRS complexes) the electrocardiogram has advanced our understanding of the cardiac arrythmias P waves are usually best seen in Lead II or in the right sided chest leads (V1 or CR1) and these leads are therefore most valuable in the disorders of cardiac rhythm

In health the heartbeat is initiated in the sinoauricular node (pace maker) which lies near the entry of the superior vena cava into the right auricle The impulse spreads through both auricles and thence to the auriculo-ventricular node lying in the inter auricular septum The AV node is continuous with the Bundle of His and its branches The commonest disorders are —

1 **Sinus tachycardia (Fig 23a)** —The cardiac impulse arises nor

mally and the electrocardiogram is normal in form The pulse



Fig 23a —Sinus tachycardia
Pulse te 120 Timing mark on this and bseq nt lectrocard ograms are t 0 2-sec intervals.

rate is increased above 90 or 100 (adults) Sinus tachycardia may result from emotion exercise fever hyperthyroidism and anæmia



Fig 23b —Sinus bradycardia
Pulse ra e 55

2 Sinus bradycardia (Fig 23b) —Again the electrocardiogram is normal in form but the heart rate is less than 60 per minute Sinus bradycardia occurs in athletes and in patients with increased intra cranial pressure myxœdema and jaundice

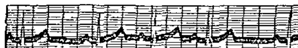


Fig 24 —Sinus arrhythmia

3 Sinus arrhythmia (Fig 24) —The cardiac impulse arises normally in the sinoauricular node the rhythmicity of which varies the heart rate increases with inspiration and diminishes with expiration The electrocardiogram is normal apart from variation in the R P intervals This arrhythmia is a normal finding in young people it is increased by deep breathing and abolished by exercise

4 Extrasystoles (Fig 25a and b) arise from irritable foci in the auricles or ventricles which stimulate the heart before the next beat is due In ventricular extrasystoles P waves are absent and the QRS complexes are like those of bundle branch block the T wave

pointing in the opposite direction to the major deflection of the QRS An extrasystole arising in the right ventricle produces

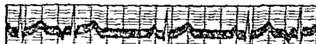


Fig 25a —Auricular extrasystoles

Note the abnormal (inverted) P wave The R–R interval is longer following the extrasystole than in the next normal cycle



Fig 25b —Ventricular extrasystoles

Note that there is no P wave before the second extrasystole and there is an abnormal P wave just before the first extrasystole

a QRS complex like that of left bundle branch block and vice versa The rhythm of the sinoauricular node is not disturbed The extrasystole comes prematurely and is followed by a pause (the compensatory pause) so that the interval between the preceding normal QRS and the following normal QRS is twice the length of the normal cardiac cycle

The electrocardiogram of an auricular extrasystole shows the P wave to be abnormal in form but the QRS which follows it is usually normal The pause which follows the extrasystole is longer than normal

Extrasystoles are thus premature beats followed by an abnormally long pause and can be recognized by auscultation or from palpation of the radial pulse Extrasystoles occur both in normal patients and in those with heart disease If an extrasystole follows after each normal beat as in Fig 25b the pulse is said to be coupled (pulsus bigeminus) Digitalis often causes coupling

5 **Paroxysmal auricular tachycardia** (Fig 26) is due to the presence of an irritable focus in the auricle which beats regularly at a rate of 150-220/min The P waves are abnormal in shape especially

when compared with normal P waves between attacks The QRS complexes are usually normal



Fig 26 —Auricular tachycardia

D ing tt k—p lse ra 5 Aft t k—si t hycard —pul t 130

Clinically this arrhythmia is characterized by its paroxysmal character and by the regular ventricular rate which is uninfluenced by exercise and respiration Paroxysms can often be terminated by pressure on the carotid sinus



Fig 27a —Auricular flutter 2 1 block

A n l t bo t 300 N t two p k d fl tt w to ea h ic l mpl

6 Auricular flutter (Fig 27a) has a similar mechanism but the auricular rate is now more rapid at 200–380/min and there is an auriculo ventricular block of 2 1 3 1 or 4 1 The pulse is regular and uninfluenced by exercise or respiration although irregularities may arise from change in the degree of auriculo ventricular block e g from 3 to 1 to 2 to 1 and back again

Auricular flutter and tachycardia may occur in hearts which are otherwise normal in thyrotoxicosis and in rheumatic or ischæmic heart disease

7 Auricular fibrillation (Fig 27b)—The irritable focus in the auricle now gives impulses at a rate of 400–600/min and there is no effective and co ordinated contraction of the auricle Because of the rapid auricular rate a physiological auriculo ventricular block develops and only occasional impulses reach the ventricle The electrocardiogram shows f (fibrillation) waves representing the auricular activity instead of P waves especially in leads CR1 or V1 The QRS complexes are normal but are irregularly spaced

Auricular fibrillation is recognized clinically by complete irregularity of the pulse both in rate and volume The latter occurs because with the irregular ventricular rate diastole may sometimes be too short to allow complete filling of the ventricles Some beats are not strong enough to open the aortic valves and hence



Fig 27b —Auricular fibrillation
N t f w d irreg l nt ul r hythm

there is a deficit between the heart rate measured by auscultation at the apex and the pulse rate at the wrist (the pulse deficit) Sometimes it is difficult to distinguish between slow auricular fibrillation and sinus rhythm or a pulse which is irregular from extrasystoles If the patient is well enough exercise makes the pulse of auricular fibrillation more irregular whereas extrasystoles tend to disappear Rheumatic heart disease ischæmic heart disease and thyrotoxicosis are the commonest causes of fibrillation



Fig 28a —Partial heart block
P R int rv l = 0 42 sec



Fig 28b —Complete heart block
A ricul r t 55 Ve t ic lar rat 39

8 Heart block (Fig 28a and b) —This may be partial or complete In the former the P-R interval exceeds 0 22 sec Sometimes the

ventricle responds only to every second third or fourth auricular stimulus In complete heart block the auricles and ventricles beat independently The ventricular rate is slow and regular (usually 20–40/min) and is controlled by a pacemaker in the auriculo ventricular node or Bundle of His The electrocardiogram shows P waves and QRS complexes which occur regularly but quite independently There is no constant P–R interval If the pace maker is in the auriculo ventricular node the QRS complexes are normal in form whereas they show bundle branch block pattern if the pacemaker is in a bundle branch

Complete auriculo ventricular block is characterized by a slow regular pulse rate The *a* waves in the venous pulse in the neck occur independently of ventricular activity seen in the carotid pulse

When the auricle contracts against closed auriculo ventricular valves venous cannon waves are seen Auscultation shows variations in the intensity of the first heart sound and in some patients auricular sounds may be heard The heart rate is little influenced by exercise

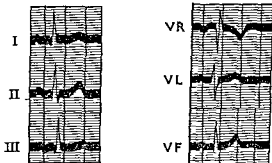
Partial heart block occurs in active rheumatic fever diphtheria and digitalis overdosage Complete heart block occurs in ischæmic heart disease and rarely in syphilis and rheumatic fever It may also be congenital when the ventricular rate is usually more rapid

9 **Ventricular fibrillation** arises from an irritable focus in the ventricle The electrocardiogram shows grossly deformed QRS complexes occurring at a rapid rate It is usually a terminal event

THE ELECTROCARDIOGRAM IN OTHER CONDITIONS

The electrocardiogram can give valuable information in conditions other than the arrythmias The following account is not intended to be exhaustive and contains many simplifications Considerable experience is required even to be familiar with the limits of normal It will be noted that many electrocardiographic abnormalities can arise from a variety of causes The interpretation of an abnormal electrocardiogram depends on the recognition of one of the basic abnormal patterns to be described below and its correlation with the clinical findings

For routine purposes the standard leads and three chest leads e g V1 V4 and V6 are recorded CR leads are sometimes used instead of V leads and the tracings are similar except that the former



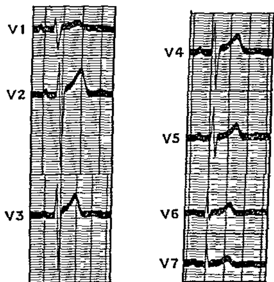


Fig 29 —Normal electrocardiogram

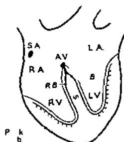
Standa d lead (I II III) V (nipolar) l mb lead V (unipol r) chest le ds V1 V7

are more pos t ve ı e the P R and T waves are taller Th s s because the voltage of a CR lead equals that of the correspond ng V lead m nus the voltage of un polar lead VR As the latter s predom nantly negat ve CR leads are more pos tıve than V leads

NORMAL PATTERNS

A normal electrocard ogram ıs shown n F g 29 The follow ng remarks apply to the standard lımb leads and to the V chest leads

The P wave n a normal tracıng ıs always upr ght except ın Lead III and occas onally ın Leads V1 V2 and V3 The P R nterval measured from the beg nn ng of the P wave to the beg nnıng of the QRS complex ıs 0 1–0 2? sec The Q wa e never exceeds 2 mm ın depth except ın Lead III where ıt may be consp cuous and ın lead V1 ın ch ldren The T wa e s always upr ght except n Lead III and Leads V1 V? and V3 ın young subjects The Q–T nterval measured between the beg nnıng of the QRS complex and the end of the T wa e represents the t me of ventr cular ex c tat on and recovery and s ın ersely proport onal to the heart rate In certa n cond t ons the Q T nterval s prolonged and tables have been p e pared show ng the max mum no mal Q T nterval at a g ven heart rate A mathemat cal formula has been de duced to correct the Q–T ınterval for the heart rate and the corrected nterval s then called the Q Tc



F g 30 —The conduct on system of the heart

SA = ın od AV = ulo- de R.B = rıgh B d His L.B = ft B d f His

4 G b g

The general fo m of the QRS com plex n the chest leads whıch can be cons dered as fac ng towards the r ght ventr cle (V1 V2 and V3) and those fa ng the left ventr cle (V5 V6 and V7) can be worked out s mply The ventr cles are act ated from the Bundle of H s and ıts branches which run close under the endocard um (F g 30) The ıntervent cular septum ıs act vated from branches derıved from the left bundle The chest leads are so connected that a wave of elec tr cal activ ty com ng towards the explorıng chest electrode pro duces an upward (pos t ve) deflect on ın the electrocard ogram and

a wave of activity going away from the chest electrode produces a downward (negative) deflection When the wave of activity has passed the trace returns to the base line again Activity occurring directly under the electrode *produces a greater deflection than* that occurring further away

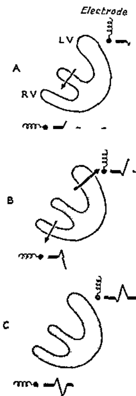


Fig 31 —To show the spread of stimulus through normal ventricular muscle

The tracings f ch t l d f c ng the r ght m icle nd of che t l d f cing th left entricle are h w The haded areas sh w st m l t d muscl The arrows show the direct n i which the umulus preads through the musl

Below are shown diagrammatically the ventricular muscle and the position of the unipolar electrodes facing towards the right ventricle and towards the left ventricle The ventricular muscle is stimulated through the Bundle of His and its branches and the septum is activated first and from its left side When for example an electrode facing the right ventricle (V1 2 and 3) is considered the initial wave of activity sweeps across the septum from left to right i e towards the electrode and there is an upward deflection in the electrocardiogram (Fig 31 A) The main muscle mass of both ventricles is excited from the endocardium towards the surface Thus the wave of activity in the right ventricle approaches the electrode while that in the left ventricle recedes from it Although the right ventricle is closer to the electrode the left ventricle is a much larger muscle mass and the electrocardiogram records a *downward deflection (Fig 31 B)* which returns to the base line as the activity fades (Fig 31 C)

Similarly in the case of electrodes facing the left ventricle (V5 6

and 7) activation of the septum produces an initial downward deflection (Fig 31 A) This is small, because the septum is at some distance from the electrode Activation of the main muscle mass of the ventricle now produces a large upward deflection (Fig 31 B) which returns to the base line as the activity fades (Fig 31 C)

ELECTROCARDIOGRAPHIC ABNORMALITIES

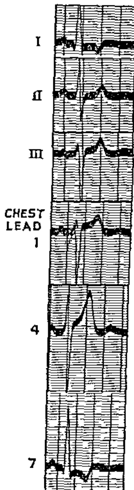
1 **Ventricular preponderance** (ventricular hypertrophy) —In ventricular preponderance the wave of activity takes longer to penetrate the hypertrophied ventricle and the electrical changes produced are greater

Left ventricular preponderance (Fig 32A) —As would be expected the S wave is larger in leads facing the right ventricle and the R wave is larger in the leads facing the left ventricle In addition there is inversion of the T waves and depression of the ST segments in limb lead I (and often II) and in the chest leads facing the left ventricle

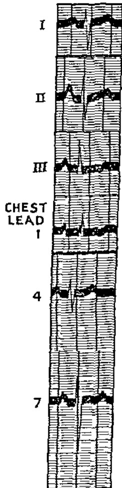
Right ventricular preponderance (Fig 32B) —The electrical changes produced by the right ventricle may now equal those produced by the left The R wave is tall in the leads facing the right ventricle Because of the delay in excitation of the right ventricle and the larger electrical changes produced there is an S wave in the leads facing the left ventricle In addition there is T wave inversion and ST depression in limb Leads II and III and in the chest leads facing the right ventricle

Often right and left axis deviation which are indicated in the limb leads are associated with right or left ventricular preponderance In left axis deviation there is a tall R in lead I and a deep S in Lead III In right axis deviation there is a deep S in lead I and a tall R in lead III Axis deviation occurs with change in the position of the heart (for example the electrocardiogram in a short obese individual in whom the heart tends to lie horizontally often shows left axis deviation) and does not necessarily indicate ventricular hypertrophy as do the changes of ventricular preponderance

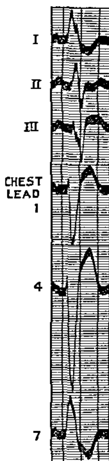
2 **Bundle branch block** (Fig 33) —In this condition the affected heart muscle is excited by transmission through the muscle itself and not through a breach of the Bundle of His Conduction through muscle is much slower than through the specialized conducting tissues and hence the QRS complex is wider than normal (i e greater than 0 12 sec) in bundle branch block

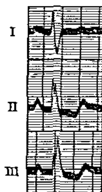


I
II
III
CHEST
LEAD
1
4
7



I
II
III
CHEST
LEAD
1
4
7





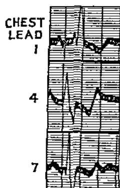


Fig 33

Left bundle branch block

N t wid (0 15 se) QRS compl es d d p w d l rr d R w h t lead 7 Thi case typ l th t th re is tS d t Q w n h t le d 1

Right bundle branch block

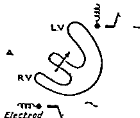
N te w d (0 15 sec) QRS comp sR p tt rn hest l d 1 and deep wid S w hest lead 7

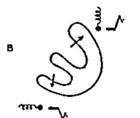
Left bundle branch block —The septum is now activated from its right side and not the left as happens normally Thus there is initially a Q wave in leads facing the right ventricle and an R wave in leads facing the left ventricle (Fig 34 A) Next the right ventricle is excited through its undamaged bundle branch and produces an R wave in leads facing the right ventricle and a smaller S wave in the more distant leads facing the left ventricle (Fig 34 B) Before this can develop fully the left ventricle is excited and causes an S wave in leads facing the right ventricle and an R^1 in those facing the left ventricle (Fig 34 C)

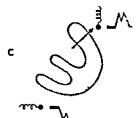
Right bundle branch block —The septum is excited normally giving an R wave in leads facing the right ventricle and a small Q wave in leads facing the left ventricle (Fig 35 A) Excitation of the left ventricle produces an S wave in leads facing the right ventricle and an R wave in those facing the left ventricle (Fig 35 B) Excitation of the right ventricle now gives an R^1 wave in leads facing the right ventricle and an S in those facing the left ventricle (Fig 35 C) The T wave usually points in a direction opposite to the major deflection of the QRS In the limb leads the major deflection of the QRS is usually upright in Lead I and downward in Lead III in left bundle branch block In right bundle branch block there is usually a deep wide S in Lead I and a tall wide R^1 in Lead III Established left bundle branch block is usually due to ischæmic heart disease Right bundle branch block occurs in some normal patients in patients with atrial septal defect (a form of congenital heart disease) and ischæmic heart disease

3 Cardiac infarction may alter the electrocardiogram by the production of abnormal Q waves or abnormalities in the S-T segments and T waves or both If an area of muscle is dead it acts as an electrical window and the activity of the muscle on the opposite wall of the ventricle is seen by the electrode and causes a Q wave in the electrocardiogram (Fig 36) S-T and T wave changes are attributed to muscle injury Within a few hours of infarction the S-T segment becomes elevated in the leads involved (Pardee s sign) (Fig 37 B) In a few days the T waves become inverted often steeply so (Fig 37 C) The S-T segment gradually returns to the base line taking several weeks to do so T wave inversion may eventually return to normal (Fig 37 D) but some

LEFT BUNDLE BRANCH BLOCK







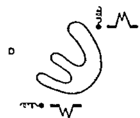
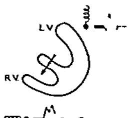
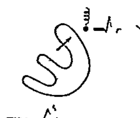
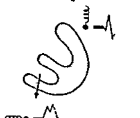


Fig 34 —Diagrams to show the spread of the stimulus through the ventricular muscle in a case of left bundle branch block

RIGHT BUNDLE BRANCH BLOCK







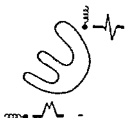


Fig 35 —Diagrams to show the spread of the stimulus through the ventricular muscle in a case of right bundle branch block

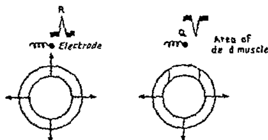


Fig 36 —Diagram to illustrate the concept of the electrical window ' and the production of Q waves in cardiac infarction

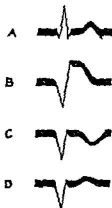
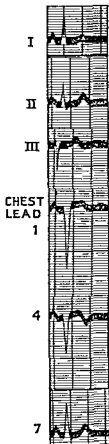


Fig 37 —Diagram to show the evolution of changes in the QRS complex S–T segment and *T* wave after cardiac infarction

A Norm l p ttern.
B A f w hou s aft r infarct on A Q wa e s p sent The S–T segment is elevated (P rdee s sign)
C. Aft t me the S–T segment turns to the base l ne a d the T w ve becomes steeply nverted.
D Aft a furth r period th T w e becomes les inve t d flat nd fin lly upright Note that th Q w e persists.

inversion usually persists Abnormal Q waves are permanent. The leads showing *Q* waves or *S–T* and *T* wave change are determined by the site of infarct The electrocardiograms illustrating classical anterior and posterior infarction (Fig 38a and b) are tracings taken several weeks after the infarction



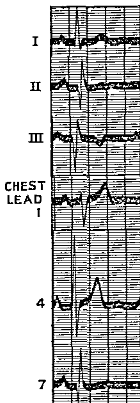


Fig 38

a Anterior cardiac infarction

N t Q w h t lead 1 and 4 T w low in le d 1 d d ph in h t l d 4

b Posterior cardiac infarction

N t Q w in l d II a d III Th T w l ghtly rt d l d II d in rted i l d III Ch st l d 7 l o h w a Q w d low T w so th t th re lly po tero-la l inf n.

Anterior infarction causes Q waves or S–T segment and T wave changes or both in limb lead I and in the chest leads facing the right ventricle

Posterior infarction causes Q waves or S–T segment and T wave changes or both in limb lead III and usually lead II as well

The changes of classical anterior and posterior infarction are not always produced for instance changes in the leads facing the left ventricle as well as those of posterior infarction would indicate postero lateral infarction In addition close familiarity with the limits of the normal electrocardiogram may allow the diagnosis of cardiac infarction to be made on less evidence than that described above

4 **P wave changes** —In right auricular hypertrophy due for example to chronic cor pulmonale or pulmonary stenosis the P wave is tall and sharp In left auricular hypertrophy especially in mitral stenosis the P wave is bifid Bifid P waves are sometimes seen in the normal electrocardiogram

5 **S–T segment and T wave changes** occur in many conditions

1 Digitalis depresses the S–T segment in all leads especially in limb leads I and II and in the chest leads facing the left ventricle

2 Flat or inverted T waves occur in myxœdema and are associated with low voltage QRS complexes and bradycardia

3 Hypokalæmia causes depression of the S–T segments and inversion of the T waves while hyperkalæmia causes tall sharp T waves

4 Pericarditis causes the S–T segment to be elevated in some leads especially in Lead II The segment retains its normal concavity This change is due to epicardial damage and is strongly suggestive of acute pericarditis In chronic pericarditis there is T wave inversion

6 **Prolongation of the Q–T interval** occurs in most cases of active rheumatic fever Often the P–R interval is also prolonged Digitalis shortens the Q–T interval

IX. X RAY EXAMINATION OF THE HEART AND AORTA

The methods in routine use are

1 **Cardioscopy** or screening which is the best single method for clinical purposes The patient is placed between the X ray tube and the screen and is usually examined in three positions

(*a*) The antero-posterior position in which the patient stands facing the observer with the shoulders parallel to the screen (Fig 39)
(*b*) The right oblique position (first oblique) in which the patient is half turned to the left with the right shoulder nearest the screen (Fig 40)
(*c*) The left oblique position (second oblique) in which the patient is half turned to the right with the left shoulder nearest the screen (Fig 41)

The chief points to note are (1) the position of the heart in the chest (2) the shape of the heart (3) its size and (4) the pulsation of the heart and aorta and any abnormal pulsation.

2 **Radiography** —Screening has the disadvantage that owing to the nearness of the tube to the patient the image seen on the screen (or photographed) is enlarged and even distorted owing to the divergence of the X rays as they traverse the chest To overcome this difficulty X ray films are taken with the tube 6 ft away as at this distance the rays are nearly parallel The radiogram so obtained gives an accurate record of the size and shape of the heart Some typical cardiac outlines are shown in Fig 42

THE NORMAL CARDIAC OUTLINE (Fig 39)

The heart is seen as a flask shaped shadow lying between the translucent lungs about one third of its area to the right and two thirds to the left of the mid line The apex of the heart is internal to the midclavicular line

The *right border* of the cardiac shadow is formed from above downwards by two curves —

(i) A slightly curved portion the outer edge of the superior vena cava with the ascending arch of the aorta

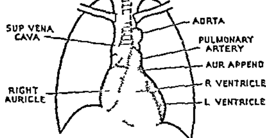


Fig 39 —The cardiac silhouette in the antero-posterior position

P t l ly eful in studying the outflow f om l ft entr cle th p lmo ry a te y nd the o t c knuckle

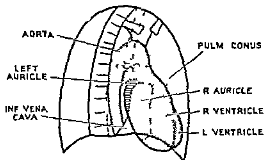


Fig 40 —The cardiac silhouette in the right oblique position

Th s posit n p rtic l ly sef l in st dy g th left tri m p lmonary conus fi st p t of rt a d bar m fille l oesophagu

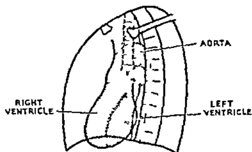
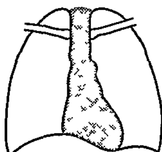


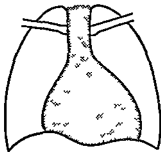
Fig 41 —The cardiac silhouette in the left oblique position

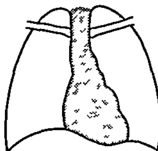
P rtic la ly useful fo studying the l ft t xl the a rtic arch a d th desce d ng a rta

(ii) A more convex portion the outer border of the right auricle which ends at the diaphragm

On the *left border* are four step-like convexities of which the second and third may be difficult to distinguish







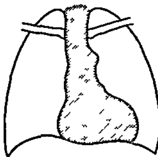


Fig 42 —Some typical cardiac outlines

A. Normal B Per ardial ff n. C M ral st s D Ao t mpet e.

(i) Above the prominent knuckle produced by the arch of the aorta as it passes backwards slightly to the left then downwards

(ii) The straighter line of the pulmonary artery

(iii) The conus of the right ventricle
(iv) Below the wide sweep of the left ventricle ending at the apex where it rests on the diaphragm

COMMON ALTERATIONS IN DISEASE

1 **Position of the heart in the chest** —Displacement of the heart as a whole i well seen in pleural effusion pneumothorax and in fibroid lung In distension of the stomach and obesity the heart is raised with the diaphragm and the apex tilted upwards The common type of scoliosis (convexity of the curve to the right) is a frequent cause of displacement of the heart to the left In narrow chests the heart often hangs mesially and seems small and slender

2 **Shape and size of the heart** —(*a*) Prominence and undue con vexity of the left ventricle is best seen in aortic incompetence aortic stenosis and cases with high blood pressure In its typical form the outline may be described as boot shaped (Fig 42D) An enlarged left ventricle is well seen in the left oblique view when it *extends backwards to cover partly the shadow of the spine*

(*b*) The left auricle is prominent in mitral stenosis and in mitral incompetence It shows in the antero-posterior view as a straight ening or convexity in the normally concave left border of the heart

Watching the progress of a thick barium paste down the œsopha gus is a valuable aid in the diagnosis of disease of the heart and greater vessels In its thoracic course the œsophagus is impressed by four structures namely the aortic arch left bronchus left auricle and descending aorta and abnormality of these causes alteration of the natural impressions In mitral stenosis with enlargement of the left auricle the depression is deepened and displaced to the right This is best seen in the right oblique view

(*c*) The outline is large and often globular when all the chambers of the heart are enlarged It is seen therefore in advanced mitral stenosis with auricular fibrillation and in combined aortic and mitral disease

3 **Shape and size of the aorta and superior vena cava** —Aortic enlargement occurs in syphilitic aortitis with aneurysm Unfolding of the aorta is seen in atheroma especially when hypertension is

associated The shadow of the superior vena cava is widened in congestive heart failure

4 **Pulsation of the heart and aorta** —An abnormal degree of aortic pulsation is a feature in the cardioscopic examination of cases of aortic incompetence In mitral incompetence the shadow of an enlarged left auricle is seen in the antero posterior view to expand markedly during ventricular systole A saccular aneurysm of the aorta appears as a rounded outgrowth from some part of the aorta which itself will often be dilated Its relation to the course of the aorta and its pulsation help to distinguish it from an intra thoracic tumour

5 **The hilar shadows and lung fields** —The hilar shadows are heavy and ill defined in cases of mitral stenosis especially in heart failure and in cases of left ventricular failure from any cause The vascular markings in the lung fields are inconspicuous in Fallot s tetralogy where much of the right ventricular output by passes the pulmonary circulation Evidence of widespread pulmonary fibrosis or infiltration lends support to a diagnosis of cor pulmonale

CARDIAC CATHETERIZATION

A flexible radio opaque catheter is introduced into an antecubital vein and followed under fluoroscopic control into the right auricle It may be pushed further into the right ventricle and the pulmonary artery The catheter can be used in the following ways

1 Blood samples can be taken from various sites for gas analysis For example—a much higher blood oxygen in a sample taken from the right auricle compared with one taken from the superior vena cava would suggest a shunt of blood from the left auricle to the right auricle

2 Pressures can be recorded at various sites For example—there is a sudden drop in pressure as the catheter is passed from the right ventricle through a stenosed pulmonary valve into the pulmonary artery

3 The catheter itself can be X rayed Its position within the heart or great vessels and its relation to other structures can be examined For example—with a large pericardial effusion the tip of the catheter in the right heart cannot be brought near the edge of the cardiac silhouette

ANGIOCARDIOGRAPHY

By the intravenous injection of a contrast medium combined with rapid serial radiography it is possible to study the heart chambers and great vessels during life. Advances in cardiac surgery have demanded precise diagnosis before operation. Angiocardiography helps to determine the site and extent of a coarctation of the aorta and hence the practicability of its resection. The investigation is also used in selecting those cases of congenital cyanotic heart disease which are suitable for operation.

CHAPTER V

CLINICAL EXAMINATION OF THE BLOOD

1 ENUMERATION OF RED BLOOD CORPUSCLES

THE instruments necessary consist of a graduated mixing pipette (Fig 43) and a counting slide Cleanse the lobe of the patient's ear with ether and dry it Make a puncture on the lower border of the lobe with an ordinary surgical needle The lobe should be pricked with a sudden stab and the blood must flow freely On no account must the blood be *squeezed* out as it is then always diluted by tissue fluid Slowly suck up blood with the pipette till the mark 0 5 is reached If one should go a little beyond the 0 5 mark the point of the pipette should be dabbed once or twice on the finger till the blood is back at the 0 5 mark Remove any surplus blood from the point taking care that the column within the pipette does not move and plunge it at once into the diluting fluid (Appendix 10) which should be standing ready in a small wide necked bottle Suck up the diluting fluid as far as the mark 101 While this is being done the pipette should be gently rotated to start the mixing Grip the pipette firmly by its end between the forefinger and thumb and shake thoroughly for at least one minute This ensures a thorough mixing of the blood with the fluid The column of diluting fluid which occupies the capillary part of the pipette does not enter into the mixture Hence if blood is sucked up to 0 5 the solution produced is in the proportion of 1 in 200 The diluting fluid in the capillary tube should now be blown out

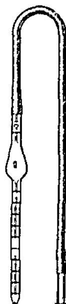


Fig 43.—Hæmocytometer pipette

The counting chamber consists of a thick glass slide with a transverse bar at its centre

the surface of which is sunk $\frac{1}{10}$ mm. below that of the slide. The bar is separated from the remainder of the slide by two transverse grooves running parallel to it, one on each side and is divided at its centre by a further groove, so that two preparations may be set up at the same time The surface of the bar

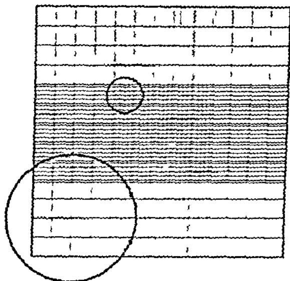


Fig 44 —Hæmocytometer slide Neubauer ruling

The small circle shows the view seen under the $\frac{1}{6}$th objective, used in counting red cells. The cells present in 5 sets of 16 small squares (marked off by double ruling) are counted. This number of cells multiplied by 10,000 equals the number of red cells per c.mm. of blood.
The large circle shows the view under the $\frac{2}{3}$rd objective used in counting white cells. The cells present in the *four large squares* (each made up of 16 smaller squares) at the four corners of the ruling are counted. This number of cells multiplied by 50 equals the number of white cells per c.mm. of blood.

is ruled with two sets of small squares, each small square having an area of $\frac{1}{400}$ *sq mm.* A specially ground thick cover-glass (ordinary cover-slips must not be used) is applied to the glass slide over the bar If the slide and cover-glass are clean and properly applied concentric colour (Newtonian) rings can be seen when one is looking almost horizontally along the surface of the

cover-glass The space left between the under surface of the cover glass and the upper surface of the bar is then exactly $\frac{1}{10}$ mm. in depth.

When the hæmocytometer pipette has been thoroughly shaken a few drops of the contents of the bulb are blown out and discarded. The pipette is then held at an angle of about 45 degrees to the surface of the counting chamber and its point applied to the

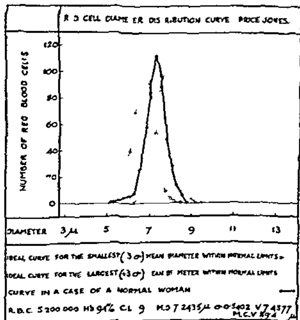


Fig 45 —Price Jones curve in a normal woman

narrow slit between the counting chamber and the cover-slip The fluid runs under the cover-slip by capillary attraction. This manœuvre requires some practice Bubbles must be avoided and the fluid must exactly fill the space between the bar and the cover slip If any fluid overflows into the grooves the counting chamber and cover-slip must be cleaned and the whole operation repeated.

When the counting chamber has been successfully filled the preparation is set aside for two minutes or so for the corpuscles to settle It is then examined with the low power to see whether any air bubbles or foreign bodies are present and whether the corpuscles are distributed with fair uniformity throughout the field after which the high power (No 2 eye piece and ⅙-in objective) is

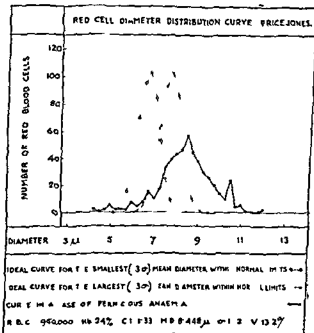


Fig 46 —Price-Jones curve in a case of pernicious anæmia

used for counting The microscope must be vertical and should be provided with a condenser and a diaphragm The light should be gradually cut off until the red cells become clearly visible Under the ⅙ in objective the little squares will be seen to be marked off into sets of sixteen by double ruling (Should the lines marking off the squares be only dimly seen it may be necessary to intensify them This is best done by rubbing the surface of the platform

with a little finely powdered graphite [the scrapings from a very soft lead pencil] and then polishing it with soft chamois leather)

At least four sets of sixteen squares should be counted under the $\frac{1}{6}$-in objective The squares in each set should be gone over systematically in horizontal rows of four at a time Of the cor puscles which lie *upon* the lines bounding the row only those on the

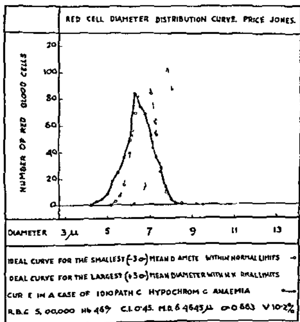


Fig 47 —Price Jones curve in a case of idiopathic hypochromic anæmia

upper and the left hand lines should be counted The number of corpuscles in each of the four sets should be approximately equal

Calculation —Count the corpuscles in each of the four horizontal rows from above downwards The total is the number of cor puscles in sixteen squares Count in this way five sets of sixteen and divide the total by eighty which gives the average of corpuscles

in one square But the dimensions of this square are $\frac{1}{400} \times \frac{1}{10}$ $= \frac{1}{4000}$ c mm Therefore if there be x corpuscles in this dimension there will be $4\,000x$ in 1 c mm But the blood was diluted 200 times Therefore in 1 c mm of blood there will be $4\,000x \times 200$ corpuscles

Suppose for example that one finds a total of 480 corpuscles in the eighty squares This gives an average of six corpuscles per square or 6 × 4 000 i e 24 000 per c mm of *diluted* blood or 4 800 000 per c mm of pure blood if the dilution was 1 in 200 Thus if a counting chamber with the Neubauer ruling (Fig 44) is used and five sets of sixteen small squares (or eighty small squares) are counted the addition of four noughts to the figure for the number of red cells in eighty small squares will give the number of red cells per c mm of blood

The normal number of red corpuscles varies within considerable limits At birth it is from 6 000 000 to 7 000 000 per c mm and falls during the first week of life to between 4 000 000 and 5 000 000 per c mm In adult life it varies in the two sexes In men the average figure is 5 500 000 per c mm The range in health is from about 5 000 000 to 6 500 000 per c mm In women the average figure is 4 800 000 per c mm and the limit in health about 4 000 000 to 5 500 000 per c mm For estimating the colour index the number five million is adopted for the sake of convenience As the error in routine single red counts is up to 500 000 differences of less than this between two counts should be ignored and preferably all counts should be expressed to the nearest half million

2 ENUMERATION OF LEUCOCYTES

A special pipette is supplied for this purpose with the Thoma Zeiss instrument It is used in the same way as the red-corpuscle pipette but the blood is less dilute The best diluting fluid is one containing 1 ml of glacial acetic acid in 100 ml of water to which enough of a watery solution of methyl green or gentian violet has been added to give the mixture a decided colour This mixture dissolves all the red cells while it stains the nuclei of the white

A large drop of blood should be allowed to exude before the pipette is filled The blood should be sucked up to the mark 0 5 the end of the pipette wiped and diluting fluid taken up to the mark 11

Owing to the relatively large calibre of the pipette the blood is apt to run out of it and so the pipette should be kept in a horizontal position as soon as it is filled with blood.

The blood and fluid are mixed as already described This produces a dilution of 1 in 20 A drop is then placed on the counting slide with the same precautions as in the case of the red cells

The leucocytes in the whole of the cross ruled area of the Thoma counting chamber which contains 400 small squares or 1 sq mm should be counted Once one has learned to distinguish leucocytes from other objects this can be done under the $\frac{2}{3}$-in objective Several preparations should be made and the total counts averaged unless one has a special counting chamber (e g Neubauer ruling Fig 44) which provides several blocks of 1 sq mm The calculation is made as follows The number of white cells counted in one sq mm is that present in $\frac{1}{10}$ c mm of the diluted fluid This figure multiplied by 200 gives the white cells per c mm of blood

In leukæmia where a very large excess of leucocytes is present one can easily count the red and the white cells in the same drop For this purpose a 3 per cent solution of sodium citrate just coloured with gentian violet is to be preferred for diluting the blood This stains the nuclei of the whites and at the same time preserves the reds Toisson s solution *(Appendix 15) may be used similarly* The dilution and calculation are the same as for the red cells

After use the diluting pipettes should be thoroughly cleaned They should be washed out (1) with distilled water (2) with absolute alcohol and (3) with ether A stream of air should then be blown through till the glass ball in the chamber moves freely without tending to adhere to the sides A few drops of anti formin sucked into the pipette quickly disintegrate any organic matter left behind Coagulated blood may be removed from the capillary tube with a horse hair If the blood adheres firmly to the pipette it may be removed by repeated rinsing with strong alkali or acid or if necessary digested away with pepsin

The **number of leucocytes in normal blood** is about 7 000 per c mm in the adult The number varies within considerable limits even in health and counts of from 3 000 to 12 000 are not necessarily abnormal In early childhood much higher numbers are reached (*see* p 388)

An absolute increase in polymorphonuclear leucocytes is referred to as *leucocytosis* and of lymphocytes as *lymphocytosis* Apart

from leukæmia which will be mentioned later important causes of *leucocytosis* are septicæmia and pyæmia the presence of pus or an abscess anywhere in the body any infection with pus forming organisms such as peritonitis pneumonia meningitis and tonsillitis Leucocytosis may also be found after acute hæmorrhage in amœbic hepatitis two or three days after a coronary thrombosis and occa sionally in the presence of malignant neoplasms particularly of the liver An absolute *lymphocytosis* apart from leukæmia is a much rarer finding It occurs particularly in glandular fever or infectious mononucleosis and after whooping cough

3 ENUMERATION OF PLATELETS

The blood platelets or thrombocytes are elliptical or circular bodies with basic cytoplasm and azurophil granules The normal platelet-count is 250 000 to 500 000 per c mm and the normal platelet ratio that is the ratio of platelets to red cells is 1 in 18 **Thrombocytopenia** or diminution of the platelets is associated with bleeding into the skin and from the mucous membranes It occurs in certain forms of purpura **Thrombocythæmia** or increase of the blood platelets may be accompanied by vascular thromboses It is sometimes encountered after splenectomy

To count the platelets the skin of the ear is cleaned with ether and on the clean surface is deposited a large drop of diluent (3 per cent sodium citrate in normal saline) The skin is then stabbed with a sharp sterile needle so that the blood oozes directly into the diluent This prevents clumping of the platelets With a platinum loop of 3 mm diameter some of the diluted blood is transferred to a slide and carefully covered with a cover slip The amount taken should be sufficient to spread out evenly between the slide and cover-slip without causing the latter to float The preparation is ringed with petroleum jelly Using a microscope fitted with moving stage square eye piece and $\frac{1}{12}$-in oil immersion objective the number of platelets and red cells is counted in several fields thus determining the ratio of platelets to red cells Knowing the red cell count the actual number of platelets per c mm is calculated

4 ESTIMATION OF HÆMOGLOBIN

Accurate hæmoglobinometry is now carried out by photo electric methods which eliminate errors due to matching colours with the eye Their accuracy is of the order ± 2 per cent For

ordinary clinical purposes Haldane s and Sahli s modifications of the original Gower s dilution and colour matching method are convenient

1 **Haldane s hæmoglobinometer** —This method uses as a standard of comparison a glass tube containing carboxy hæmoglobin Recent work has shown that 100 per cent on the scale corresponds to 14 8 grm. of hæmoglobin per 100 ml The colour of the standard is fairly permanent if it is kept in the dark

The method is as follows —

0 4 per cent ammonia solution (4 ml Liqu ammon sp gr 0 880 in a litre of distilled water) is placed in the diluting tube up to the 20 mark Water may be used if no ammonia is available and if the patient is very anæmic the tube should be filled only to the 5 or 10 mark An adequate puncture is then made in the finger or the lobe of an ear and the capillary pipette provided filled to the 20 c mm mark without delay The skin and the pipette must be perfectly clean and dry The pipette should be held horizontally the point dabbed on the observer s finger till the column of blood is exactly at the 20 mark and then wiped clean of any surplus blood The blood is then gently blown into the diluting tube where it should sink without mixing appreciably with the fluid in the tube the pipette is washed out several times with this fluid and withdrawn The solution is then saturated with carbon monoxide with a capillary pipette or lumbar puncture needle connected by rubber tubing to the gas mains The gas may be bubbled through the solution or passed over it while the tube is slanted to expose the greatest fluid surface to the gas Distilled water is now added drop by drop from a drop bottle or pipette After each addition the diluting tube is held between the thumb and forefinger and inverted to ensure thorough mixing Any fluid left on the thumb must be wiped back into the tube Water is added in this manner until the tint in the diluting and standard tubes appears identical The tubes should be matched in daylight (but not direct sunlight) against a northern sky or against white paper at an angle of 45 or against an electric bulb viewed through an opal glass screen It is absolutely necessary to transfer the two tubes repeatedly while they are being matched or avoidable error will be made

The level on the graduated tube is read one minute after the last

drop of distilled water was added The observation is repeated after the addition of another drop and if necessary another until the point is reached when the tints are again just unequal and the average of the two readings is accepted as the final result

Even among skilled observers the error of this method may be considerable Given careful attention to the details of technique and a good eye for matching tints the error should not exceed 5 per cent In practice a difference of 5 per cent. between two observations by the same observer will usually indicate a real change in the patient rather than an error in the method and a difference of 10 per cent will nearly always do so Differences of less than 5 per cent between two observations by the same observer and of less than 10 per cent between two observations by different observers should be disregarded

2 **Sahli s hæmoglobinometer** —This method employs as a standard of comparison a glass tube containing acid hæmatin the hæmoglobin in the diluting tube being converted to the same sub tance by the addition of hydrochloric acid In some models a perma nent coloured glass standard is employed The method can be used in places where no coal gas is available

Instruments can be obtained calibrated so that 100 per cent is equiva lent to 14 grm hæmoglobin per 100 ml when the reading is made 5 minutes after the addition of the blood to the acid in the diluting tube

The method is as follows —

The diluting tube is filled to the 10 or 20 mark with decinormal hydrochloric acid 20 c mm of blood are taken up with a pipette as described above and blown gently into the tube the pipette being rinsed out several times with the acid solution The tube is allowed to stand for exactly five minutes Then adding water and matching the tints is carried out as with the Haldane instrument except that the fluid in the tube is stirred with a small glass rod after each addition of water instead of the tube being inverted

The error of this method is not less than that of the Haldane and the same precautions should be used in interpreting the results recorded The difference between the Haldane scale (100 per cent = 14 8 grm) and the scale of the Sahli instruments (100 per cent = 14 0 grm) may be neglected It goes without saying however that any report on hæmoglobin percentage *should state what is* meant by 100 per cent in terms of grm per 100 ml.

The normal hæmoglobin varies within considerable limits and differs in the two sexes and at different ages The mean hæmoglobin of healthy men may be taken as 15 6 grm per 100 ml equivalent to 113 per cent in the Haldane scale and the normal range from about 100 to 120 per cent The mean figure for healthy women may be taken as 13 7 grm per 100 ml equivalent to 99 per cent on the Haldane scale and the normal range from about 90 to 110 per cent At birth the hæmoglobin may be 140 to 150 per cent it falls during the first few weeks to a level of 70 per cent. or 80 per cent and increases slowly during infancy and childhood

From the red cell count and the percentage of hæmoglobin the colour index can be calculated This expresses the mean hæmoglobin content of a single cell as compared with that of an arbitrary normal cell It is obtained by dividing the hæmoglobin expressed as a percentage of normal (100 per cent = 14 5 grm of hæmoglobin) by the red cell count expressed as a percentage of normal (assumed for convenience to be 5 000 000) Thus if the hæmoglobin is 40 per cent and the red cell count is 4 000 000 the colour index is

$$40 - \left(\frac{4}{5} \times 100\right) = \frac{40 \times 5}{4 \times 100} = 0\ 5$$

More simply it is calculated by doubling the first two figures of the red cell count and dividing the product into the figure for the percentage of hæmoglobin The normal colour index by definition is 1 0 but figures from 0 85 to 1 15 are not necessarily abnormal The colour index is useful as it divides anæmias approximately into three classes those with a high colour index including pernicious and similar anæmias those with a colour index about unity such as anæmia following a recent large hæmorrhage and those with a low colour index such as the iron deficiency anæmias

For accurate hæmatological diagnosis the colour index has however the disadvantages that it is based on arbitrary standards of normal and that it takes no account of cell size For this reason certain absolute values are often employed

1 **Volume of packed cells (V P C or Hæmatocrit)**

This is determined by centrifuging uncoagulated blood in a graduated tube until the corpuscles are packed down to a constant

volume The volume of packed cells is then expressed as a percentage of the original column of blood 5 ml of blood from a vein is placed in a tube containing 4 mg solid potassium oxalate and 6 mg solid ammonium oxalate (with this salt mixture there is no shrinkage of cells) A Wintrobe s hæmatocrit tube (which can also be used for determining the sedimentation rate p 159) is then filled to the 100 mark with this blood with a capillary pipette and centrifuged at 2 500 revolutions per minute for 30 minutes The original column of blood in the tube being 100 mm long the volume of packed cells can be read directly as a percentage Given the red cell count in millions per c mm the hæmoglobin expressed in grm per 100 ml and the volume of packed cells the following additional absolute values can then be calculated

2 **Mean corpuscular volume (M C V)**

$$\text{M C V in cub } \mu = \frac{\text{Volume of packed cells in ml per 1 000 ml of blood}}{\text{Red cells in millions per c mm}}$$

3 **Mean corpuscular hæmoglobin (M C H)**

$$\text{M C H in micro-micrograms } (\gamma\gamma) = \frac{\text{Hb in grm per 1 000 ml of blood}}{\text{Red cells in millions per c mm}}$$

4 **Mean corpuscular hæmoglobin concentration (M C H C)**

M C H C %

$$= \frac{\text{Hb in grm per 100 ml of blood}}{\text{Volume packed red cells in c mm per 100 c mm of blood}} \times 100$$

The mean normal and range of normal of these values are as follows —

	Mean normal	*Range of normal*
M C V in cub μ	86	78-94
M C H in $\gamma\gamma$	29 5	27-32
M C H C. %	34	32-38

Of these values the most useful are the M C V and M C H C If the M C.V is above 94 cub μ the anæmia is macrocytic in type if it is between 78 and 94 the anæmia is normocytic and if it is below 78 cub μ the anæmia is microcytic The M C H C expresses

the amount of hæmoglobin per 100 ml of cells rather than the amount per 100 ml of blood and is therefore the true measure of the degree of saturation of the cells with hæmoglobin It cannot be increased for red cells cannot hold a greater concentration of hæmoglobin than the normal When it is reduced below 32 per cent it is an indication that the patient should be treated with iron

If the mean corpuscular diameter M C D is also determined the mean red cell thickness can be calculated This is important in acholuric jaundice in which the M C T is increased The M C D can be determined by the use of an halometer or more accurately by the method of Price Jones

RED CELL DIAMETER DISTRIBUTION CURVE (PRICE JONES)

Variations in the diameters of red blood cells are observed and measured in dried films These are dried in air without heat fixed and stained with Jenner stain for two minutes and after washing with distilled water and drying are super stained with a weak aqueous solution of eosin for two minutes

A projection apparatus adjusted for a magnification of 1 000 diameters is then arranged to project the microscope field on to a sheet of paper Five hundred red cells are then outlined in pencil Two diameters maximum and minimum of each of these cells are then measured to 0 5 *mm with a glass millimetre scale and can be expressed directly in* terms of μ The average of these two measurements is taken as the mean diameter of the cell expressed to the nearest 0 25 μ When 500 cells have been measured a diameter distribution curve is plotted showing the number of red cells of a given mean diameter at intervals of 0 25 μ and the mean corpuscular diameter (M C D) the standard deviation (σ) and the coefficient of variability (v) are calculated

Fig 45 shows the normal mean diameter of 500 red cells in a healthy woman to be 7 2 μ To the right of this is the ideal curve for the largest mean diameter within normal limits (7 7 μ) to the left the ideal curve for the smallest mean diameter within normal limits (6 7 μ) A mean diameter outside these limits is abnormal By counting the cells that fall outside these limits it is possible to calculate the degree of macrocytosis or microcytosis The healthy range of the coefficient of variability which gives mathematical expression to the degree of anisocytosis present is from 5 3 per cent to 7 3 per cent with a mean of 6 3 per cent (Price Jones)

In pernicious anæmia the average mean diameter is high (8 3 μ) So also is the average coefficient of variability (13 0 per cent) Price Jones concludes that a high variability is almost more constant though less characteristic than a high mean diameter In the case illustrated here

the mean diameter is 8 45 μ and the co fficient of variability is 13 2 per cent The high variability is to be seen in the wide base of the curve (Fig 46) It is a mathematical expression of a high degree of anisocytosis The megalocytosis is to be seen in the fact that the peak of the curve falls outside the right hand boundary of the calculated curve

In *idiopathic hypo hromic anæmia the mean diam ter may* be less than normal but often falls within normal limits In a series of eight cases Price Jones found the mean diameter ranged from 6 2 μ to 6 7 μ In all there was a microcytosis 6 to 37 per cent of the cells lying outside the left hand boundary of the calculated curve In the case illustrated here (Fig 47) *the mean diameter is* 6 5 μ

5 MICROSCOPICAL EXAMINATION OF THE BLOOD

Films are usually made on slides The slides should be of colourless glass thin and with ground edges they should be entirely *free from grease To ensure this they should be dropped one by one into an enamelled iron dish containing 10 per cent chromic acid* and boiled for 20 minutes They should then be tipped into a shallow basin and water allowed to run on them till the washings are colourless After this they are covered with spirit and finally transferred with forceps to a wide necked stoppered bottle con taining absolute alcohol When required they should be picked *out with forceps* excess of alcohol drained and the slide dried off with a clean cloth They should finally be rubbed with a clean handkerchief Slides may also be satisfactorily cleaned by polish ing them with the finest emery paper and then leaving in absolute alcohol until required

If slides have to be cleaned in a hurry glacial acetic acid followed by water and alcohol gives good results

How to make films —Apply one end of a slide to a drop of blood and place the slide on a level surface holding it with the thumb and index finger of the left hand The narrow edge of a *second slide* is placed in the drop and held there till the blood has spread *across* it it is then drawn slowly over the whole length of *the first slide* The inclination of the second slide to the first should be 45 and there should be no pressure whatever between *the two surfaces* The more slowly *one* slide is drawn over the other the thinner is the resulting *film* Smooth spreading of the film is aided by

warming the first slide in the flame of a spirit lamp before applying it to the drop of blood After the blood is spread it should be dried by being waved rapidly in the air to prevent undue shrinkage of the cells

How to stain the film —Either of the two following methods gives excellent results —

1 *Jenner s stain* —(The stain consists of a 0 5 per cent solution of a specially prepared crystalline compound of methylene blue and eosin in pure methyl alcohol) Films are made in the usual way As soon as they are dry a few drops of the solution are poured on and they are covered with watch glasses to prevent evaporation and precipitation of the stain Pour off in one to four minutes Rinse in *distilled* water till pink (five to ten seconds) Dry rapidly high over a flame or by waving in the air Mount *in xylol balsam In a successful film the red corpuscles are brown* ish red nuclei are blue platelets mauve the granules of poly nuclear cells and myelocytes red basophils dark violet bacteria filarial and malaria parasites blue

2 *Leishman s stain* —This is a simplification of the method of staining first introduced by Romanowsky The dry film is well covered with the stain which should be evenly distributed over the entire slide or cover glass At the end of one minute double the quantity of distilled water is carefully added and mixed with the stain At the end of seven minutes the mixture is poured off and the film covered with distilled water for two minutes The water is then washed off with fresh distilled water and the film gently blotted dry with clean blotting paper When dry it can be mounted in xylol balsam or examined directly under the oil immersion lens

Examination of the film —In a good film the cells are evenly spread and there are no rouleaux After a general examination of the film to see whether red cells white cells and platelets are present in about normal proportions to observe the shape size and staining properties of the red cells and to see whether primitive or abnormal red or white cells are present a **differential count** is made to ascertain the relative numbers of the different varieties of leucocyte 200-500 cells must be counted

It is often necessary also to calculate the *absolute* number of each kind of white cell per c mm of blood as otherwise a relative

increase or diminution of one kind may be mistaken for an absolute increase or reduction Throughout *adult life the absolute number* of polynuclears per c mm is about 4 000 whilst that of the lymphocytes is about 2 000

The following are the varieties of leucocytes found in normal blood (Plate 13) with their relative proportions —

1 *Neutrophil polymorphonuclear* —Cells with multilobed nucleus and fine neutrophil or faintly oxyphil granules 60–65 per cent

2 *Eosinophil polymorphonuclears* —Cells with multilobed nucleus and coarse strongly oxyphil granules 2–3 per cent

3 *Basophil polymorphonuclears* (or mast cells) —Cells with very pale cytoplasm a nucleus usually bilobed and coarse basophil granules 0–0 5 per cent

4 *Monocytes* —Cells with a characteristic notched or kidney shaped nucleus and a slightly basophilic faintly reticular cytoplasm 3–5 per cent

5 *Large lymphocytes* with round nucleus and clear basophilic cytoplasm 5–10 per cent

6 *Small lymphocytes* with round deeply staining nucleus which almost fills the cell leaving a rim of strongly basophilic cytoplasm 20–25 per cent

The changes in the relative proportion of these cells in leucocytosis have been mentioned (p 139)

In the lymphatic form of leukæmia there is an enormous increase in the number of the lymphocytes (Plate 16)

In the chronic myeloid form of the disease the neutrophils eosinophils and basophils are all increased and in addition bone marrow cells—myelocytes—appear in the blood These are often of large size with a single round nucleu and contain granules which may be either neutrophilic or eosinophilic in reaction (Plate 16)

A relative diminution of the leucocytes is spoken of as leucopenia As a rule in leucopenia the diminution affects chiefly the granulocytes hence the name granulocytopenia There is of course a relative lymphocytosis Leucopenia occurs in —

(1) Infections such as typhoid fever undulant fever and measles
(2) Exhaustion of the bone marrow as in aplastic anæmia
(3) *Crowding out of the leucopoietic tissues* by abnormal erythropoiesis as in pernicious anæmia and acholuric jaundice

(4) Sensitivity to many drugs e g sulphonamides thiouracil amidopyrine troxidone

The red cells may present various alterations in disease (Plates 14 and 15) The alterations may affect—

(*a*) Their *size* and *shape* Instead of the normal sized erythrocytes small cells may appear devoid of the usual central indentation (microcytes) or unusually large forms may be met with (macrocytes) particularly in pernicious anæmia Instead of being rounded the corpuscles may become oval pear shaped etc These changes are spoken of collectively as *poikilocytosis*

(*b*) The *staining power* of the cells may be altered Thus instead of taking up eosin in the normal manner they may stain with the basic dye and have a violet or even bluish tinge This is spoken of as *polychromatophilia* It is seen in various kinds of anæmia

The cells may also take up the basic stain in a stippled manner This is known as *punctate basophilia* Slight degrees occur in anæmias and a considerable degree may be found in persons exposed to lead

(*c*) *Nucleated forms* may appear *Normoblasts* can be distinguished from lymphocytes (for which at the first glance they are apt to be mistaken) by (1) the more homogeneous and intense staining of the nucleus (2) the presence round the nucleus of a cell body which stains red (3) their smoother contour

Megaloblasts are large nucleated red corpuscles They have a relatively small and characteristically stippled nucleus and a large cell body which always exhibits polychromatophilia Megaloblasts are a characteristic but rare feature of the blood in pernicious anæmia

Reticulated red corpuscles or reticulocytes can be demonstrated by *supravital staining* that is by the application to fresh blood of a special dye before the use of a fixative The reticulocytes are the youngest red cells in the circulation and in the normal blood rarely exceed 2 per cent of the total red cells In circumstances demanding active regeneration of blood they may reach 15 to 30 per cent The reticulocyte is of slightly larger diameter than the red cell and contains a delicate cytoplasmic network which later disintegrates and disappears as the cell matures into a red blood corpuscle

The best dye to use for supravital staining of the blood is brilliant cresyl blue Saturate 0 85 per cent sodium chloride solution with the dye filter through a double filter paper and centrifugalize Pour off the supernatant dye solution and keep it in a stock bottle For use dilute a small quantity with four volumes of 2 per cent sodium citrate in physiological saline Puncture the ear and draw a large drop of blood into a Wright s pipette Follow up the blood by an equal volume of dye Blow out on to a slide mix thoroughly take up again into the Wright s pipette seal off and incubate at body temperature for 20 minutes Then make films by the same technique as for blood films and stain them with Jenner or Leishman in the usual manner The reticulocytes present among at least 500 red cells should be counted

Parasites in the blood —These may be looked for in fresh blood under a cover slip in which some of them e g microfilariæ and trypanosomes may be seen alive and moving or in fixed and stained films which may be thick or thin The making of thin films has already been described (p 146)

Fresh films —The slides and cover slips must be clean and free from grease and should be held by the edges so that the fingers do not touch their surfaces Prick the finger or lobe of the ear and wipe away the first drop of blood When a second has accumulated just touch it with the centre of a cover slip and lower the latter carefully on to the centre of a slide The blood spreads out into a film If a drop of blood of the right size has been used the centre of this film is almost colourless If the centre is red apply a little pressure The film should be examined with the light well cut down in turn under the $\frac{2}{3}$ $\frac{1}{6}$ and if necessary $\frac{1}{12}$ objectives

Thick films —The thick film method enables a much larger amount of blood to be examined in a given time than is possible with thin films and is therefore valuable for the detection of parasites when these are present in small numbers Take a drop of blood on to the centre of a slide and spread it with a triangular needle until print can be clearly seen through it while it is still wet Allow the film to dry thoroughly by leaving it protected from dust for a minimum of two hours and if possible overnight If speed is essential it may be dried by warming very cautiously When the film is thoroughly dry it must be dehæmoglobinized by immersing

the slide gently in an upright position in a beaker of clean and preferably distilled water and allowing the hæmoglobin to dissolve out. Gentle movement helps but if the slide is handled roughly the film may become detached In about five minutes when the film is colourless and opaque it is removed from the water and allowed to dry Thereafter it can be stained with Jenner s or Leishman s stain in the same manner as a thin film

Another method uses a dilute watery solution of Giemsa s stain which dehæmoglobinizes and stains the film in one operation The slide with the film facing downwards is placed across two thin glass rods in a flat glass dish and a freshly prepared dilute watery solution of Giemsa s stain (1 drop of stain to each ml of water) is run under the slide and left for 15 to 20 minutes The slide is then washed gently in a dish of water and allowed to dry in a semi vertical position Thick films should be inspected rapidly under the ⅓-in objective and then examined systematically under the oil immersion

Recognition of parasites —The important parasites of the blood are the parasites of malaria microfilariæ of several varieties try panosomes Leishman Donovan bodies and the spirochætes of relapsing fever

For the diagnosis of malaria thick films should be used for the detection of parasites and thin films for their identification Films should preferably be taken when the patient s temperature is raised. Thick films stained as described should be examined systematically for 10 to 15 minutes before it is concluded that no parasites are present The recognition of parasites in thick films requires prac tice White cells platelets bacteria, the remains of reticulocytes and miscellaneous dirt can be mistaken for parasites Parasites have definite morphological and staining properties and objects which do not show these are not parasites

For details of the identification of the different types of parasites in thin films larger works must be consulted The main distin guishing points are as follows (Plate 17) In infection with *plasmodium falciparum* which produces malignant tertian malaria schizogony generally takes place in the tissues so that except in rare cases in moribund patients ring forms and a few crescent shaped gametocytes only are seen in the peripheral red blood cells Ring forms of any species consist of a rim of cytoplasm which stains

blue and a small nucleus or chromatin dot which stains red The rings of plasmodium falciparum are usually though not invariably small and delicate and the red cells are not enlarged More than one ring may appear in a single red cell and some rings may have two chromatin dots Marginal forms or formes appliquees with the parasite lying along the edge of the cell may be seen A few crescent shaped gametocytes which are easily recognized (Plate 17 13 14) may be seen in films from untreated patients but if absent at this time they may appear particularly in patients treated with mepacrine some seven to ten days after the beginning of treatment

In the remaining three species schizogony takes place in the peripheral blood so that *ring forms large trophozoites* and schizonts will be present together in films

In infections with **plasmodium vivax** which produces benign tertian malaria the rings are large and stout and often measure one third of the diameter of the red cell The red cells may be enlarged and if properly stained may show well marked Schuffner s dots (Plate 17 1 2) Large irregular trophozoites containing brown pigment and mature schizonts with 16 or more merozoites may be seen (Plate 17 3)

In infections with **plasmodium malariæ** which produces quartan malaria the ring forms are also large and stout but the larger trophozoites are more compact and dense looking and frequently take a characteristic band form (Plate 17 6 7) Mature schizonts contain some eight merozoites arranged in a rosette form around a mass of pigment Further Schuffner s dots are not seen and the red cells are not enlarged

In infection with **plasmodium ovale** which is much the rarest of the four species the parasites have some of the characteristics of plasmodium vivax (e g large and prominent Schuffner s dots) and others of plasmodium malariæ (e g compact large trophozoites and occasional band forms) The most characteristic feature is the distortion in shape of the red cells which become oval or fimbriated and the schizonts only contain 8 to 10 merozoites (Plate 17 9 10)

In some cases mixed infections may be present

In the diagnosis of **trypanosomiasis** examination of the blood is generally less efficient than the examination of fluid obtained by gland puncture An enlarged gland usually in the posterior

triangle of the neck is held firmly between the thumb and fingers of the left hand while a moderate sized hypodermic needle is plunged through the skin and into the substance of the gland. A small amount of gland fluid passes into the needle and suction is neither necessary nor desirable. The needle is withdrawn and its contents blown out on to clean glass slides. The fluid should be examined fresh and unstained as described for fresh blood films, and thin films should be stained with Leishman's stain.

Trypanosomes (Plate 17, 20) may also be found in thick or thin blood films and, in advanced cases, in films made from the deposit of centrifuged cerebro-spinal fluid. For methods of concentrating trypanosomes in the blood larger works must be consulted.

The important trypanosomes of man are *Trypanosoma gambiense* and *rhodesiense* which cause African sleeping sickness and, as seen in human blood, are usually morphologically indistinguishable; and *Trypanosoma cruzi* which causes Brazilian trypanosomiasis. The latter exists chiefly in a non-flagellated form in the organs and muscles, and only occasionally appears in the blood as a flagellate trypanosome (Plate 17, 17).

In a suspected case of trypanosomiasis several specimens of gland fluid and of blood should be examined, both fresh and unstained and in stained films. Fresh films should be examined under the $\frac{2}{3}$ and then under the $\frac{1}{6}$ objective. Under the latter trypanosomes may be seen "lashing" their way amongst the cells and are often first detected by the commotion they produce in the latter. This movement must not be confused with that produced by the *Treponema* of relapsing fever.

In stained films, examined under the oil immersion lens, typical trypanosomes are seen as elongated fusiform structures some 14 to 30 µ long and 1 to 3 µ broad with a longitudinal undulating membrane and a terminal flagellum projecting from the anterior end (Plate 17, 17). There is a centrally placed nucleus and at the posterior end a smaller black-staining kinetoplast. The shorter forms are more stumpy and may have little or no free flagellum.

In the diagnosis of **Kala-azar** Leishman-Donovan bodies may be looked for in the blood or in material obtained by sternal, gland, spleen or liver puncture. Of these examinations of marrow obtained by sternal puncture (p. 163) is probably the simplest and safest method, but in occasional cases the parasites may be found by the examination of stained blood films when they are seen in

PLATE 13

Blood cells norma and abnormal

The left hand column represents cells produced in the bone marrow and present in the blood in disease only The central column represents normal and abnormal varieties of red cells The right hand column illustrates the normal leucocytes of the blood

(L hman ta n)


N t ph l y loc t
l g type


N rm l red ll


P lym rphon cl
t ph l


N t ph l my loc t
(m ll t pe)


P kilocyt


I os ph l


N ph l p l yt


P l h mat ph l


B soph l


E ph l locyt


N rmobl


M t


Basoph l my locyt


M g l bl t


Larg l m phoc t


P t t
b soph l


Sm ll lymphocyt

Plate 13

PLATE 13

Blood cells norma and abnormal

The left hand column represents cells produced in the bone marrow and present in the blood in disease only The central column represents normal and abnormal varieties of red cells The right hand column illustrates the normal leucocytes of the blood

(Le hman s sta n)

PLATE 14

a The blood in iron-deficiency (hypochromic microcytic) anæmia

Note 1 The red cells are small (microcytic) and poorly stained (hypo chromic)

2 There is some anisocytosis (undue inequality in size) and poikilocytosis (irregularity in shape)

3 The film also shows one polymorphonuclear leucocyte one small lymphocyte and three groups of platelets

b The blood in addisonian or pernicious anæmia

Note 1 The red cells are large (macrocytic) and some are unusually deeply stained (hyperchromic)

2 There is considerable anisocytosis and some poikilocytosis

3 The film also shows one small lymphocyte one normoblast and a group of platelets

The red cells in Plate 15b may be taken as normal for purposes of comparison

(D wn by I Kr m)

the cytoplasm of large mononuclear cells When direct microscopic examination fails culture methods for which larger works must be consulted are frequently successful

The parasites may be seen in thick or thin stained blood films which should be searched systematically under the oil immersion objective The parasites are seen as round or oval bodies from 2 to 5 μ in diameter containing a large round or oval solid looking nucleus and a smaller more deeply stained and usually rod shaped kinetoplast In Leishman stained films the cytoplasm is blue and the nucleus and kinetoplast are red (Plate 17 16)

While the term Leishman Donovan bodies strictly applies to the Indian form of kala azar exactly similar Leishmania may be found in the blood or tissues in cases of Mediterranean kala azar from fluid obtained by puncture at the margin of the lesion in the various forms of cutaneous leishmaniasis or tropical sore and from the mucous membranes of the mouth nose or throat in espundia or South American leishmaniasis

Adult filarial worms or macrofilariæ are parasites of the lymphatic system or connective tissues Their presence is diagnosed by the finding of their larvæ or microfilariæ in the blood stream Three main varieties of microfilariæ are found in the blood of man These are (1) **Filaria bancrofti** which is found in the blood stream in any numbers only at night and which causes filariasis characterized by irregular fever lymphangitis and various forms of elephantiasis (2) **Filaria loa loa** which is found in the blood stream only by day and causes loiasis characterized by transient red painful swellings known as Calabar swellings and (3) **Filaria perstans** which is *non periodic* appearing equally by day and night and has no recognized pathogenic effects

If filariasis is suspected blood should be examined say at 8 a m noon and 4 p m and again at 8 p m midnight and 4 a m Fresh unstained films should be used for the detection of the filariæ and stained ones thick if the larvæ are scanty and thin if they are plentiful for their identification

In fresh unstained films microfilariæ are easily seen under the ⅔ objective as actively moving linear objects In stained films they are seen as wormlike objects with a round head and a pointed tail from 5 to 8 μ broad (i e about the diameter of a red cell) and from 100 to 300 μ long (Plate 17 19) The main differentiating features of the three species apart from their periodicity

are as follows *Filaria bancrofti* and *F loa loa* have a delicate sheath which can be seen where it projects beyond the rounded head and pointed tail of the larva whereas *F perstans* is unsheathed All larvæ have a central column of nuclei extending from the head more or less to the tail In *F loa loa* and *perstans* the nuclei extend to the extreme tip of the tail whereas in *F bancrofti* the column ends short of the tip

Microfilariæ do not stain well by Leishman s stain and a better staining method is as follows Thick films should be dehæmaglobinized as described under malaria and allowed to dry There after the method is the same for thick or thin ones The films are fixed for five minutes with methyl alcohol and rinsed in water The slides are then flooded with hæmatoxylin—preferably Ehrlich s mixture though Mayer s hæmalum can also be used—which is gently warmed until steam rises and an occasional bubble is seen The heat is withdrawn for a few minutes and then reapplied the process being repeated several times in a period of fifteen minutes If the slide shows signs of drying additional stain must be added When cool, the slide is immersed in water to wash off the stain and then allowed to stand in running tap water until a blue colour appears generally in about quarter of an hour When quite dry it should be mounted in Canada balsam Microfilariæ are readily recognized by the intense staining of their nuclei and the sheath if present, is easily seen

The **Treponema recurrentis** of relapsing fever should be sought under the ⅙ objective in fresh unstained films of blood and in thin films stained by Leishman s method under the oil immersion

Fresh unstained films should be examined with the light well cut down. Agitation of the red cells usually calls attention to the presence of parasites which may otherwise be difficult to detect

In thin stained films the spirochæte is seen as a linear object with tapering ends 0 4 μ in breadth and 10 to 30 μ in length The spiral shape which it possesses in life is lost and the body lies in irregular curves In searching for the organism it is important to direct the eyes deliberately to the spaces between the red cells rather than to the red cells or the parasite may be missed (Plate 17 18)

6 SPECTROSCOPIC EXAMINATION

The diagnosis of suspected carbon monoxide poisoning may be established by spectroscopic examination Some blood is obtained by pricking the thumb and squeezing two or three drops into several ml of distilled water The solution has a cherry red colour Place some in a thin flat glass tube and examine with a hand spectroscope Direct the instrument as in all such examinations towards a white cloud and not towards the sun Two bands (Plate 18) are seen (bands of carboxyhæmoglobin) occupying very much the same position as the oxyhæmoglobin bands They are distinguished from the latter by the fact that the addition of a few drops of ammonium sulphide produces no alteration in them

7 ESTIMATION OF COAGULATION TIME

The coagulability of the blood can be estimated with a fair degree of accuracy by means of Wright s coagulometer The instrument consists of a series of fine tubes of equal calibre which are kept immersed in water at body temperature Blood is drawn into each of the tubes at definite intervals and after the lapse of varying periods of time one blows down the tubes in succession If the blood can no longer be blown out coagulation has occurred The interval between the filling of the tube and the occurrence of coagulation is known as the *coagulation time*

At a temperature of 37 C the coagulation time of a healthy individual is about four minutes It is increased in hæmophilia but normal in thrombocytopenic purpura It is also increased during the administration of anticoagulants

ESTIMATION OF THE BLEEDING TIME

The bleeding time is determined by stabbing the ear with a sharp sterile needle and blotting off the drop of blood every 30 seconds until it ceases to ooze from the puncture The blots are recorded in series along a strip of blotting paper and subsequently counted The normal bleeding time is 4 to 5 minutes It is increased in thrombocytopenic purpura but normal in hæmophilia

ESTIMATION OF PROTHROMBIN INDEX

The concentration of prothrombin in the plasma is estimated by measuring the length of time taken for the plasma to clot in

the presence of an excess of thrombokinase and of calcium ions Blood is withdrawn into an oxalate solution which prevents it clotting by removing free calcium ions and centrifuged To the plasma thromboplastin—Russell viper venom or an extract of brain tissue—is added to provide an excess of thrombokinase Calcium chloride is then added and the time taken for the plasma to clot measured with a stop watch This is normally 25 to 30 seconds Several determinations on normal subjects are made at the same time The prothrombin index equals

$$\frac{\text{Average normal time} \times 100}{\text{Observed clotting time of specimen tested}}$$

The prothrombin index is reduced when insufficient vitamin K is absorbed in liver disease in new born infants with melæna neonatorum and during the administration of synthetic anti coagulant substances which interfere with the formation of pro thrombin

8 SEDIMENTATION RATE

In health the red cells of the blood agglutinate very little on standing the resultant clumps of corpuscles are small and sedi ment slowly On the other hand in certain diseases the red cells agglutinate into larger clumps which sediment more rapidly As a sick person improves the clumps become smaller and the sedimen tation rate slower There is no standard method for estimating the erythrocyte sedimentation rate The Westergren method is simplest and the one most generally in use 0 2 ml of 3 8 per cent sodium citrate is sucked into a 1 ml syringe and blood is then drawn by venepuncture up to the 1 ml mark The mixture is shaken so that it is evenly mixed The blood is then sucked into a pipette 2 5 mm in diameter and graduated to 200 mm This is fixed in a stand and sealed by being clipped on to a cork at the bottom The distance settled in millimetres in one hour is the sedimentation rate The normal is 1 to 7 mm the slightly abnor mal range is 8 to 15 mm and the grossly abnormal 15 to 110 mm or even higher

In the Wintrobe method a hæmatocrit tube (as used for deter mining the volume of packed cells p 144) is filled to the 100 mark with oxalated blood and allowed to stand vertically for one hour

PLATE 15

a The blood in acholuric jaundice

Note 1 The red cells are about normal in size

2 Some of them have a diffuse bluish coloration (polychromatophilia) others are slightly smaller in diameter than normal and unusually densely stained (spherocytes)

3 The film also shows a polymorphonuclear leucocyte a normoblast and three groups of platelets

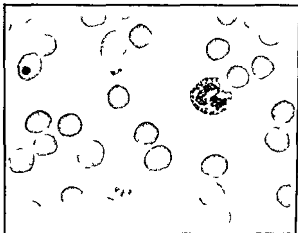
The red cells on Plate 15b below may be taken as normal for purposes of comparison

b The blood in acute leukæmia

Note 1 The red cells are normal in appearance

2 The film shows five myeloblasts and two neutrophil proleucocytes

(Drawn by I Kr m r)



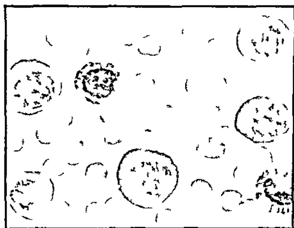
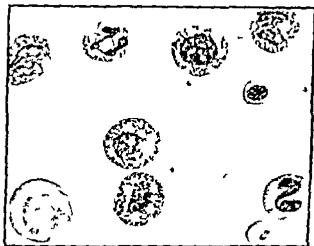


Plate 15



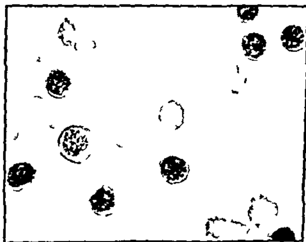


Plate 16

PLATE 16

a The blood in chronic myeloid leukæmia

Note 1 The red cells show slight anisocytosis and poikilocytosis

2 The white cells shown include one myeloblast three neutrophil myelocytes and four neutrophil polymorphonuclear leucocytes of which two are immature with only two lobes each The remaining white cell is a small lymphocyte

b The blood in chronic lymphatic leukæmia
(small-cell type)

Note 1 The red cells show some anisocytosis and poikilocytosis

2 The white cells present consist of eight small and one large lympho cyte some with nuclei of slightly primitive appearance

3 Three smudge cells are also shown

(Drawn by I K m)

By this method the normal sedimentation for men after one hour is 0 to 9 mm and for women 0 to 20 mm The advantage of this method is that the hæmatocrit tube can subsequently be centrifuged to determine the volume of packed cells and the sedimentation rate corrected for the degree of anæmia present This can be done by means of a chart specially prepared for the purpose (*vide American Journal of Medical Science* Vol 189 1935 p 102)

The sedimentation rate is reduced in polycythæmia and in congestive heart failure and increased in anæmia in all but the mildest toxic and infective conditions and in many cases of cancer especially in carcinomatosis It is also increased in pregnancy normal or abnormal It rarely has any specific diagnostic value but indicates the presence of a disease process of some sort which may be anything from a cold to a carcinoma It is excessively high in carcinomatosis multiple myeloma and disseminated lupus erythematosis It is of some use in distinguishing between organic and functional disorders for in the presence of a raised sedimentation rate a diagnosis of a functional disorder should never be made until all possible investigations to exclude an organic cause have been undertaken Conversely a low edimentation rate does *not* necessarily exclude organic disease

The sedimentation rate is also useful in following the progress of patients suffering from chronic diseases It is especially useful in chronic pulmonary tuberculosis rheumatoid arthritis and acute rheumatism As long as the sedimentation rate is raised there is an active disease process and this is frequently found a considerable time after the temperature has returned to normal It is convenient to estimate the sedimentation rate at weekly intervals A trend towards acceleration of the rate is an indication that the disease process is active whereas a trend towards slowing of the rate indicates that it is subsiding

9 FRAGILITY OF THE RED CORPUSCLES

The fragility of the red blood-corpuscles is shown by their inability to resist hæmolysis in diminishing strengths of salt solution Normal salt solution is usually taken as 0 85 per cent of NaCl but normal red cells do not hæmolyse until a dilution of about 0 4 per cent. is reached In most forms of chronic jaundice the red cells are less fragile than normal cells and do not hæmolyse

in a salt solution appreciably less than 0 4 per cent In acholuric jaundice the fragility of the red cells is greatly increased and hæmolysis takes place in strengths of salt approaching that of normal saline Not uncommonly hæmolysis begins at 0 6 per cent of salt or even higher The undue fragility of the red cells is the most constant and characteristic sign of this disorder and such wide deviation from the normal is found in no other condition

The estimation of the fragility of the red blood-corpuscles is simple Two series of test tubes each containing about 5 ml of a range of dilutions of NaCl in water are put up and to each of one series added a drop of blood from the patient under investigation and a drop of normal blood to each of the other series It is not necessary to wash and centrifuge the red cells the whole blood may be taken The normal control series should never be omitted The range of dilutions should extend from 0 8 per cent to 0 2 per cent and there should be a difference of not less than 0 05 per cent between each dilution The solutions can be rapidly made by placing in one burette distilled water and in a second burette a 1 per cent solution of NaCl in water Into the first tube run 4 ml of the salt solution and 1 ml of water giving 0 8 per cent NaCl into the following test tube put ¼ ml less of salt solution and ¼ ml more of water and so on until a strength of 0 2 per cent NaCl is reached Each tube should be thoroughly shaken after the dilution is made and again after the blood has been added Finally the tubes are allowed to stand at room temperature until all the intact corpuscles have settled to the bottom The first tube showing hæmolysis is recognized by the faintly pinkish colour of the supernatant fluid

10 STERNAL PUNCTURE

Sternal puncture is employed to examine the marrow in anæmias and leukæmias to detect certain parasites e g Leishman Donovan bodies in kala azar and in the diagnosis of carcinomatosis myelo matosis and disorders of lipoid storage such as Gaucher s disease For most purposes the examination of smears of marrow aspirated through a needle is quite adequate For certain purposes the sternum needs to be trephined and a small piece of marrow excised for histological section This is essential for an accurate diagnosis of the degree of hypoplasia present in aplastic anæmias and

to establish the diagnosis in rare cases of osteosclerosis with anæmia

The apparatus used for puncturing the sternum is a stout trochar and cannula fitted with an adjustable stop and so made that a hypodermic syringe fits into the top of the cannula The skin and tissues down to the periosteum of the sternum are anæsthetized with 2 per cent novocaine without adrenalin A small incision is made in the skin with a tenotome slightly lateral to the mid point of the manubrium or over the middle of the sternum opposite the 2nd or 3rd intercostal space The stop on the trochar is unscrewed

	Average per cent	Range of normal per cent
Hæmocytoblasts	1 0	0 0– 2 0
Promyelocytes	4 0	0 7–14 0
Neutrophil myelocytes	13 0	2 0–20 0
Eosinophils	1 0	0 5– 3 0
Basophils	0 25	0 0– 0 5
Proleucocytes	29 0	10 0–48 0
Polymorphonuclear leucocytes	18 0	9 0–34 0
Total myeloid cells	66 5	
Proerythroblasts	0 5	0 0– 2 0
Normoblasts basophilic	2 0	0 3– 3 0
polychromatic	12 0	5 0–15 0
orthochromatic	5 0	1 0– 7 0
Total erythroid cells	19 5	
Lymphocytes	10 0	2 5–24 0
Monocytes	2 0	0 0– 5 0
Plasma cells	0 5	0 0– 2 0
Megakaryocytes	0 25	0 0– 2 0
Cells in mitosis	0 25	0 1– 0 4
Smudge cells		0 3– 0 2
Myeloid-erythroid ratio	3 4 1	8 1 to 2 1

Modified from *Recent Advances in Clinical Pathology* Churchill London 1947

and adjusted according to the build of the patient the adjustment may well be 1 cm at first The corticalis of the sternum is pierced with a boring motion the stop being unscrewed to lengthen the projecting portion of the trochar if necessary Some force may be required but it is easy to recognize the entry of the tip of the trochar into the marrow cavity The Salah needle commonly used in this manner was actually designed to be tapped through the corticalis of the sternum with a small mal et When the cavity of the ster num has been entered the trochar is withdrawn the hypodermic syringe inserted and as few drops as possible of red fluid aspirated Without disconnecting the syringe the cannula is removed and a swab put over the incision The plunger of the syringe is pushed and as quickly as possible films are made on slides from the fluid expressed from the cannula If marrow has been successfully aspirated the films will contain fat and appear messy and uneven They may be stained with Jenner s or Leishman s stain

For the interpretation of marrow films larger works must be consulted The normal cell count or myelogram may be taken as in the table on p 164

11 BLOOD GROUPING

Agglutination is due to the interaction of an agglutinogen (or antigen) present in red cells with an agglutinin (or antibody) present in plasma Many different agglutinogens are present in human red cells but in blood transfusion the important ones are the A and B agglutinogens of Landsteiner since these are the only ones for which the corresponding agglutinins α (anti A) and β (anti B) are normally present in human plasma It is evident that a person s plasma cannot contain the agglutinin for an agglutinogen present in his own red cells but with rare exceptions α or β (or both) agglutinins are present in the plasma whenever the red cells do not contain the corresponding agglutinogen On the basis of the agglutinogens present in their red cells human beings are divided into four groups—AB A B and O *

The agglutinogens and agglutinins present in the four groups

Th gro p AB A B d O corre p d pect l to gr p I II III d IV of th Moss lassifica io d IV II III d I f th J ky l ficat In iew f h dange ou mistak wh ch m y rise f m c f betwee th se tw ld merical l ss ficat they h ld be b d d t f th in m io l AB A B d O l ssib tio

together with the approximate percentage of individuals found in each group in England are shown in the following table —

Group	Corpuscles	Plasma	Percentage of individuals in group
AB	Agglutinogens AB	Agglutinins o	3
A	A	β	42
B	B	α	9
O	O	αβ	46

When a transfusion is given the danger is that the agglutinins of the recipient s plasma will agglutinate the donor s red cells How this may come about is shown in the next table where + indicates agglutination and — no agglutination —

Recipient's group and agglutinins present in plasma	Donor's group and agglutinogen present in cells			
	AB	*A*	*B*	*O*
AB Agglutinin o	—	—	—	—
A Agglutinin β	+	—	+	—
B Agglutinin α	+	+	—	—
O Agglutinin αβ	+	+	+	—

From this table it will be seen that the group of any blood can be determined with the aid of a supply of suitable group A and B sera For the corpuscles of the blood under test will be agglutinated by both sera if it is of group AB by group B serum only *if it is of group A by group A serum only if it is of group B* and by neither serum if it is of group O This method of grouping is known *as the indirect test In the direct test the donor s red cells* are tested with the recipient s serum a procedure which is also often referred to as cross matching

From this table it will also appear that blood may be given safely to any recipient from a donor of *the same (or homologous)* group or to any recipient from a donor of Group O (the so-called universal donor)

This is true in the great majority of cases but neglects several complications which occasionally give rise to severe and even fatal transfusion reactions in apparently compatible transfusions

1 **Groups A_1 A_2 A_1B and A_2B** —Group A is strictly divisible into A_1 accounting for $\frac{4}{5}$ of group A persons and A_2 accounting for $\frac{1}{5}$ Group AB is similarly divisible into A_1B and A_2B The practical importance of this fact is that A_2 cells react more weakly than A_1 cells with group B testing sera Hence if a low titre testing serum is used group A_2 may be mistaken for group O and group A_2B for B This will be avoided if high titre B serum is used and if typing is carefully performed

2 Anti O agglutinin is present spontaneously but rarely in A_1 B and A_1B sera It reacts with all O cells and also with 95 per cent of A_2 cells for which reason it is often called alpha$_2$ agglutinin

The extra agglutinins associated with these sub-groups are usually present in small amounts unless stimulated by a previous trans fusion Hence a single transfusion of group O blood to recipients of other groups is usually quite safe and in practice reactions due *to repeated transfusions are rare One danger of repeated trans* fusions of O blood to recipients of other groups is however that the formation of anti O agglutinins may be stimulated

3 **The Rh factor** —If red cells from a rhesus monkey are trans fused into a rabbit the rabbit s serum will subsequently cause agglutination in 85 per cent of human bloodts and no agglutination in the remaining 15 per cent 85 per cent of human bloods are therefore described as rhesus positive in hat they have an Rh agglutinogen in their red cells and 15 per cent as rhesus negative in that they have no such agglutinogen Under normal circum stances Rh negative persons have no Rh agglutinin in their plasma but the formation of this agglutinin may be stimulated either by a transfusion of Rh positive blood or in the case of Rh negative women by the presence of an Rh positive fœtus *in utero* in some cases in which the father is Rh positive If Rh positive blood is given to an Rh negative person in whom the formation of Rh agglu tinins has been stimulated in one of these ways—a severe or even fatal transfusion reaction may be produced It follows that trans fusion reactions in repeated transfusions may be due to this cause

rather than to the incompatibilities mentioned above and that Rh negative pregnant and puerperal women can only safely be transfused with Rh negative blood of correct A B O group

METHODS

Blood grouping —If a transfusion is to be given enough blood should be collected from the recipient to provide a sample of red cells for the indirect test and a sample of plasma for cross matching with the donor s red cells

The recipient s finger or ear is pricked and one drop of blood allowed to drop into a small tube containing 1 ml of a 3 per cent sodium citrate solution Blood from the same puncture is then allowed to run into a piece of capillary tubing about 1 mm diameter and 2 in long One end of this tubing is sealed and the tube then centrifuged to separate the serum or simply left standing when enough serum for cross matching purposes will usually separate out

Tube method —Into two small tubes approx 2 in by ¼ in clearly marked A and B place respectively one volume of group A typing serum and one volume of group B typing serum with the aid of capillary pipettes making sure that the same pipette is never used for both sera One volume of saline is added to each tube and then one volume of the red-cell suspension prepared as stated above The tubes are inverted to ensure thorough mixing and then left at room temperature for two hours They are then gently flicked with the finger when the presence or absence of agglutination is usually quite obvious If there is any doubt the contents of the tube can be taken up in a Pasteur pipette transferred to a slide and examined under the ⅔ objective Group AB cells are agglutinated by both A and B sera Group A cells are agglutinated by group B serum but not by group A serum Group B cells are agglutinated by group A serum but not by group B serum and group O cells are not agglutinated by either group A or group B sera

Tile method —This is less accurate than the tube method, but is quicker and requires less apparatus

Large drops of group A and group B typing sera are placed on a white porcelain tile and clearly marked A and B To each a very small drop of the patient s whole blood is added and the tile gently rocked Usually agglutination is clearly visible to the

naked eye before the fluid dries on the tile This method should only be used by persons with considerable experience of blood grouping but it is useful in an emergency

Cross matching —This should never be omitted The best method is to place one volume of the recipient s serum (removed by means of a fine pipette from the capillary tube prepared as described above) one volume of saline and one volume of a 5 per cent suspension of the donor s blood in 3 per cent citrate (obtained as described under blood grouping) into a small tube The tube is then inverted left at room temperature for two hours and examined for agglutination exactly as described under blood grouping In emergency a large drop of the patient s serum may be mixed on a slide with a drop of a 5 per cent suspension of the donor s red cells obtained as above The slide should then be left for ten minutes with occasional rocking and examined under the ⅔ objective

For further details of blood groups and methods reference must be made to larger works or to the Medical Research Council War Memorandum No 9 *The Determination of Blood Groups* published by H M Stationery Office

12 SPECIAL CHEMICAL METHODS OF BLOOD INVESTIGATION

1 **Serum bilirubin** —An excess of bilirubin in the blood may result from an increased rate of destruction of red cells (hæmolysis) or from interference with excretion due to liver damage (intra hepatic obstruction) or obstruction to the bile ducts (extra hepatic obstruction) In both these forms of obstruction bile which has passed through the liver cells may be reabsorbed into the blood stream The Van den Bergh reaction was formerly used to distinguish between hæmobilirubin (bilirubin derived from excessive hæmolysis) and cholebilirubin (bilirubin reabsorbed from liver canaliculi or bile ducts) and so to differentiate between hæmolytic and obstructive jaundice In practice this distinction is rarely difficult and the real difficulty lies in distinguishing between intra and extra hepatic obstruction in which the Van den Bergh reaction is of no help Estimations of the serum bilirubin (normal up to 0 5 mg per 100 ml) are used however to detect subclinical jaundice (0 5 to

2 0 mg per 100 ml) when no abnormal coloration may be visible and to follow the progress of cases of jaundice

2 **Estimation of urea in blood** —The *reagents required* are —

0 6 per cent acid potassium phosphate
Coarse ground soya bean
Caprylic alcohol
Anhydrous potassium carbonate
$\frac{1}{1}$ *normal hydrochloric acid*
$\frac{1}{10}$ normal sodium hydroxide

Methods —In tube B (Fig 48) place 2 ml of blood 2 ml of the acid potassium phosphate solution a knife point of the soya bean powder ground to an emulsion with about 1 ml of water and 3 drops of caprylic alcohol Incubate this tube for 20 minutes at about 40 C

Into tube C place 10 ml $\frac{N}{100}$ hydrochloric acid

Tube A contains about 20 ml of dilute sulphuric acid

When the incubation of B is complete connect the tubes as shown and connect the outlet tube of C to a strong water pump Start the current of air gently and pour about 4 grm anhydrous potassium carbonate into B reinserting the stopper at once Run a gentle current for about 10 minutes and then run for 50 minutes as fast as possible without frothing over The urea in the blood is converted into ammonium salts by an enzyme in the soya bean this ammonia does not escape as the solution is kept acid by the phosphate When the fluid in B is made strongly alkaline the ammonia is set free and carried by the air-current to be caught by the acid in C The acid in A traps any ammonia in the air After the current of air has run for an hour in all disconnect from the pump wash down the entry tube of C with distilled water inside and out and titrate the remaining acid with $\frac{N}{100}$ sodium hydroxide using methyl red as indicator

If a ml of alkali are used $(10 - a)$ ml of acid have been neutralized

Since 1 ml of $\frac{N}{100}$ acid is neutralized by 0 14 mg of ammonia nitrogen and 0 30 mg of urea contain 0 14 mg of nitrogen

then 2 ml of blood used contain $(10 - a)$ 0 30 mg of urea
100 ml $(10 - a)$ 15 mg of urea

It is preferable that the ends of the entry tubes should be blown into bulbs perforated with about 6 holes of the size that would be made by a needle

If it is suspected that the blood contains excessive amounts of urea 1 ml only should be used

This method may be used for the estimation of urea in urine it is more accurate than the hypobromite method 0 5 or 1 0 ml of urine is used 5 ml of acid phosphate solution and $\frac{N}{10}$ acid and alkali instead of $\frac{N}{100}$

Normal blood contains from 20 to 40 mg of urea per 100 ml With moderate reduction of renal efficiency normal values are usually found With more severe degrees of reduction the blood urea begins to rise and may reach 600 mg per 100 ml before

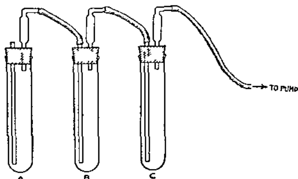


Fig 48 —Apparatus for estimation of urea in blood

(F f t t)

death Patients with blood urea above 100 mg per 100 ml not due to a temporary condition which can be relieved rarely live more than a year For methods of estimating the renal efficiency *see* p 230

3 **Estimation of sugar in blood** (1) **MacLean s method** —This method is included because it does not require a colorimeter **The solutions required are** —

(*a*) Sodium sulphate 15 per cent To this add 0 1 ml of acetic acid per 100 ml immediately before use

(*b*) Dialysed iron That supplied by the British Drug Houses is satisfactory

(*c*) Copper solution Dissolve 12 grm of potassium bicarbonate with

gentle heat in 70 ml of distilled water add 8 grm of anhydrous potassium carbonate Dissolve 0 35 grm of copper sulphate crystals in a few ml of water in another vessel and add to the bicarbonate solution without waiting for the potassium carbonate to dissolve completely When effervescence has finished complete the solution of the carbonate by heating Add 0 05 grm of potassium iodate and 0 5 grm of potassium iodide shake make up to 100 ml filter This solution should not be used for two days after this it keeps indefinitely It must be standardized *by taking 2 ml in 10 c c of acid sodium sulphate solution and adding* 2 ml of 75 per cent hydrochloric acid One minute after effervescence has subsided shake and titrate with $\frac{N}{400}$ sodium thiosulphate as described later 2 ml should require about 11 c c of thiosulphate This standardization should be repeated from time to time

(*d*) $\frac{N}{400}$ sodium thiosulphate solution made by diluting 5 ml of $\frac{N}{10}$ thiosulphate to 200 ml The thiosulphate solution should be kept in blue bottles in the dark and the $\frac{N}{10}$ solution standardized from time to time

(*e*) 1 per cent solution of soluble starch

(*f*) 75 per cent hydrochloric acid solution (75 ml concentrated hydrochloric acid diluted to 100 ml)

Rubber tubing is wound round the patient s finger to produce congestion a little powdered potassium oxalate is spread on the place to be punctured and a needle stab made in the middle of the oxalate patch just above the root of the nail 0 2 ml of blood are allowed to run into a *0 2 ml pipette which is placed in contact with the side of the drop* and held horizontally If too much blood is obtained it may be brought down to the mark by tapping on the finger nail When the blood reaches the mark, any that remains on the point is wiped away * The blood is added to 23 8 ml of acid sodium sulphate solution which has been run from a burette into a Duro glass flask and the pipette washed out by sucking up and blowing out the solution The mixture is heated until it begins to boil the flask being fitted with a rubber stopper through which passes a glass tube gradually drawn out to a capillary point This allows the escape of air and practically prevents the loss of fluid The flask is cooled under the tap and 1 ml of dialysed iron is added with constant shaking The fluid is filtered and 0 ml of filtrate used for sugar estimation or if a high blood sugar is expected 10 ml of filtrate made up to 20 ml with acid sodium sulphate

* It is ry import nt that th p pett sho ld be t rely f f om grease It ho ld therefore be wa hed out af h perim w h a hot m t re of s lph ric d nd potassi m b ch om t Afte po t th m t re it is w hed o t w th dist led wate follow d by l hol d ther d then dried w th th a d of a bl pipe and gentl h t

Two ml of solution No 3 are added to the filtrate taken and it is heated over a flame which will bring the mixture to brisk boiling in 100 secs In order to ensure a flame of the right height the arrangement in Fig 49 is used By previous trials it is found what pressure of gas as shown by the manometer will bring 20 ml of acid sulphate solution to a brisk boil in 100 secs and in subsequent work the screw clip is adjusted until the manometer records this pressure the same flask gauze tripod and burner must always be used

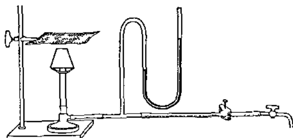


Fig 49 —Apparatus for estimation of sugar in blood

Boiling is continued for 6 minutes after the liquid begins to boil vigorously all over The flask is cooled under a tap 2 ml of 75 per cent HCl are added and it is shaken gently until effervescence has finished and at intervals for a minute after The fluid is titrated with $\frac{N}{400}$ sodium thiosulphate until the yellowish colour of iodine has almost disappeared 2 or 3 drops of soluble starch solution added and the titration continued until the blue colour disappears

The number of ml of thiosulphate used is subtracted from the number used by 2 ml of solution 3 alone and the percentage of sugar in the blood is calculated from the table Thus if the figure obtained on titration was 8 98 ml and 2 ml of solution 3 alone required 11 05 ml the difference 2 07 ml represents the sugar present From the table below 2 07 ml of thiosulphate = 0 18 mg of sugar therefore $\frac{20}{25} \times 0\ 2$ ml of blood contain 0 18 mg of sugar therefore 100 ml of blood contain 0 112 grm

In all methods of blood sugar estimation the filter papers must be free from carbohydrate Whatman s No 1 are suitable

TABLE SHOWING AMOUNT OF GLUCOSE (IN MG) AND ITS PERCENTAGE EQUIVALENT TO $\frac{N}{400}$ SODIUM THIOSULPHATE SOLUTION WHEN 20 ML OF BLOOD FILTRATE ARE USED

$\frac{N}{400}$ Thiosulphate ml.	Glucose mg	$\frac{N}{400}$ Thiosulphate ml	Glucose mg
0 12	0 03 = 0 018	2 61	0 22 = 0 137
0 25	0 04 = 0 025	2 74	0 23 = 0 143
0 38	0 05 = 0 031	2 86	0 24 = 0 150
0 50	0 06 = 0 037	2 99	0 25 = 0 156
0 62	0 07 = 0 043	3 11	0 26 = 0 162
0 73	0 08 = 0 050	3 24	0 27 0 168
0 86	0 09 = 0 056	3 36	0 28 = 0 175
0 99	0 10 = 0 062	3 49	0 29 = 0 181
1 13	0 11 = 0 068	3 61	0 30 = 0 187
1 26	0 12 = 0 075	3 74	0 31 = 0 193
1 39	0 13 = 0 081	3 87	0 32 = 0 200
1 53	0 14 = 0 086	3 99	0 33 = 0 206
1 67	0 15 = 0 093	4 12	0 34 = 0 212
1 80	0 16 = 0 100	4 24	0 35 = 0 218
1 94	0 17 = 0 106	4 37	0 36 = 0 225
2 07	0 18 = 0 112	4 49	0 37 = 0 231
2 22	0 19 = 0 118	4 62	0 38 = 0 237
2 35	0 20 = 0 125	4 74	0 39 = 0 243
2 49	0 21 = 0 131	4 87	0 40 = 0 250

(2) **Method of Folin and Wu**—This is the simplest and most rapid method if a colorimeter is available The *solutions required* are —

10 per cent sodium tungstate $\frac{2}{3}$ normal sulphuric acid Alkaline copper solution —

Dissolve 40 grm of anhydrous sodium carbonate in 400 ml of water Add 7 5 grm of tartaric acid and dissolve then add 4 5 grm of copper sulphate weighed accurately and dissolve The solution of each solid should be complete before the addition of the next Make up to 1 000 ml Test for absence of cuprous salts by mixing 2 ml with 2 ml phosphomolybdic acid reagent the deep blue colour should almost vanish

Phosphomolybdic acid reagent —

Dissolve 35 grm of molybdic acid and 5 grm of sodium tungstate in a large flask add 200 ml of 10 per cent sodium hydroxide 200 ml

of water and boil until ammonia no longer comes off Cool dilute to 350 ml and add 125 ml of 85 per cent phosphoric acid Make up to 500 ml

Stock glucose solution —

1 per cent pure glucose dissolved in 2 5 per cent benzoic acid

Weak glucose standard —

Make 1 ml of the stock glucose up to 100 ml with 2 5 per cent benzoic acid solution Contains 1 mg of glucose in 10 ml

Strong glucose standard —

Make 2 ml of the stock glucose up to 100 ml with benzoic acid solution Contains 2 mg of glucose in 10 ml

Precipitation of proteins—Place 2 ml of blood which has been prevented from clotting by the addition of a small amount of potassium oxalate in a small flask Add 14 ml of distilled water shake gently to cause hæmolysis Then add 2 ml of sodium tungstate solution and 2 ml of ⅔ normal sulphuric acid shaking during the addition Cork the flask and shake violently After 15 secs filter

Reduction of the copper solution—In a tube which has a bulb holding just under 4 ml at its end and above this a neck about ¼ in in diameter place 2 ml of the filtrate and 2 ml of the copper solution In two other tubes (I) and (II) place 2 ml of the weak (I) and strong (II) glucose solutions add 2 ml of copper solution to each Place the tubes in a boiling water bath for 6 minutes cuprous oxide is formed

Reduction of the phosphomolybdic acid reagent by the cuprous oxide and comparing the depths of colour produced—Cool the tubes in water for two minutes Add to all three 2 ml of the phosphomolybdic acid reagent Stand two minutes for the solution of the cuprous oxide to be completed a deep blue colour develops Dilute to 25 ml (it is convenient to have the tubes graduated at 25 ml) Mix well and compare the depths of colour in the tube containing the blood filtrate with that in tube (I) or (II) which it most nearly resembles in depth If tube (I) is used as standard and the reading of the standard in the colorimeter is 20 and if the unknown is a Since the depth of colour is proportional to the amount of glucose present

$$2 \text{ ml of filtrate contain } \frac{20}{a} \times \frac{2}{10} \text{ mg}$$

$$20 \text{ ml} \qquad \frac{20}{a} \times 2 \text{ mg}$$

$$2 \text{ ml of blood} \qquad \frac{20}{a} \times 2 \text{ mg}$$

$$100 \text{ ml} \qquad \frac{20}{a} \times 100 \text{ mg}$$

Plate 17

Parasites of the blood

1	*Plasmodium vivax*	Ring stage
2		Amœboid form
3		Fully developed schizont
4		Male gametocyte
5		Female gametocyte
6	*Plasmodium malariæ*	Compact form
7		Band form
8		Fully developed schizont
9	*Plasmodium ovale*	*Female gametocyte*
10		Fully developed schizont
11	*Plasmodium falicparum*	Red blood corpuscles containing multiple infections of various types of young rings
12		Old ring showing altered staining reaction and Maurer's dots
13		Male gametocyte or crescent
14		Female gametocyte or crescent
15		Pigment in polymorphonuclear leucocyte
16	*Leishmania donovani* (from a spleen smear)	Some lying free others within the cytoplasm of an endothelial cell
17	*Trypanosoma cruzi*	Adult form as seen occasionally in the blood of patients suffering from Chagas disease
18	*Treponema recurrentis*	

(Nos 1–18 magnification × 2 000 approx)

19	*M filaria loa loa*	× 600 approx
20	*Trypanosoma rhodesiense*	As seen in a thick blood film of patients suffering from trypanosomiasis × 1 000 approx

(Drawn by W Cooper)

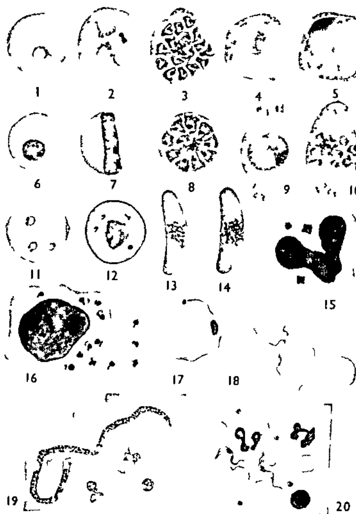


Plate 17

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Parasites of the blood

1	*Plasmodium vivax*	Ring stage
2		Amœboid form
3		Fully developed schizont
4		Male gametocyte
5		Female gametocyte
6	*Plasmodium malariæ*	Compact form
7		Band form
8		Fully developed schizont
9	*Plasmodium ovale*	Female gametocyte
10		Fully developed schizont
11	*Plasmodium falciparum*	Red blood corpuscles containing multiple infections of various types of young rings
12		Old ring showing altered staining reaction and Maurer's dots
13		Male gametocyte or crescent
14		Female gametocyte or crescent
15		Pigment in polymorphonuclear leucocyte
16	*Leishmania donovani* (from a spleen smear)	Some lying free, others within the cytoplasm of an endothelial cell
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19	*M filaria loa loa*	× 600 approx
20	*Trypanosoma rhodesiense*	As seen in a thick blood film of patients suffering from trypanosomiasis × 1 000 approx

(Drawn by W Cooper)

PLATE 18

Spectra of hæmoglobin and its derivatives compared with solar spectrum

1 Solar spectrum
2 Spectrum of dilute solution of oxyhæmoglobin
3 hæmoglobin
4 carboxy hæmoglobin
5 acid hæmatin in ethereal solution
6 alkaline hæmatin
7 methæmoglobin
8 hæmochromogen
9 acid hæmatoporphyrin

Aft r H ll b t *H db k f Phy l l gy*

If tube (II) was used for the comparison 100 ml of blood contain $\frac{40}{a} \times 100$ mg

The precipitation of the blood should be done within a few minutes after it is drawn as the glucose is destroyed on standing

The blood of normal persons taken when they have fasted for some hours contains 80 to 120 mg of glucose per 100 ml After a meal containing carbohydrate this may rise to 150 mg Usually only traces of glucose appear in the urine when the blood sugar is below 180 mg per 100 c c the renal threshold

In diabetes mellitus the blood may contain much larger quantities of glucose up to 600 mg or more

The glucose tolerance test—To determine a patient s ability to metabolize carbohydrate he is given a test dose of 50 grm of glucose after which the blood sugar is estimated and the urine tested for the presence of sugar at intervals for the next two or three hours

The usual method is as follows The patient who has been allowed no food since the previous evening has blood taken for a fasting blood sugar and empties his bladder He then drinks 50 grm of glucose dissolved in 100 ml of water Further specimens of blood are withdrawn and further samples of urine collected at the end of ¾ hour and two hours

Under normal conditions the fasting blood sugar is between 80 and 120 mg per 100 ml The blood sugar ¾ hour after the glucose is taken has risen to 140 to 170 mg per 100 ml and at the end of two hours it has fallen to its fasting level The corresponding specimens of urine contain no sugar since sugar does not pass into the urine in detectable quantities in normal persons till the blood sugar reaches 180 mg per 100 ml—the so-called renal threshold

In renal glycosuria or lowered renal threshold the blood sugar curve is normal but sugar is found in one or more of the corresponding specimens of urine

The test is also employed in the diagnosis of diabetes mellitus For this purpose it is essential that the patient should have been eating a normal amount of carbohydrate during the previous week Normal persons on a low carbohydrate diet may show abnormal blood sugar levels after a test dose of glucose and hence be mis

diagnosed as cases of mild diabetes if this precaution is not observed Definite cases of diabetes mellitus can generally be recognized by the history and examination of the urine but if the glucose tolerance test is performed the fasting blood sugar is usually well above 120 mg per 100 ml and may be 200 or 300 mg After the test dose of glucose the blood sugar rises and does not return to normal levels at the end of two hours

The significance of moderately raised blood sugar curves without symptoms of diabetes is not known Persons who have them sometimes progress to a true diabetes mellitus If a single blood sugar determination is used to confirm a diagnosis of diabetes mellitus one taken an hour after a carbohydrate meal should be employed rather than a fasting one In true diabetes mellitus the blood sugar at this time will be found to be 250 mg per 100 ml or more

CHAPTER VI

THE RESPIRATORY SYSTEM

I ANATOMICAL LANDMARKS

(Plates 8 9 10 11)

1 **Lobes of the lungs** —It is important to know the limits of the individual lobes of the lungs A line from the 2nd thoracic spine to the 6th rib in the mammary line corresponds to the upper border of the lower lobe (the major interlobar fissure) A horizontal line on the right side from the sternum at the level of the 4th costal cartilage drawn to meet the first marks the boundary between the upper and middle lobes (the minor inter lobar fissure) The greater part of each lung as seen from behind is composed of the lower lobe only the apex belonging to the upper lobe while the middle and upper lobes on the right side and the upper lobe on the left occupy most of the area in front In the axillary regions parts of all the lobes are accessible

The bifurcation of the trachea corresponds in front with the lower border of the manubrium sterni that is with the angle of Louis behind with the disc between the 4th and 5th thoracic vertebræ The median angle of the scapula is generally on a level with the disc between the 1st and 2nd thoracic vertebræ and its inferior angle with the body of the 8th thoracic vertebra The median angle of the scapula just covers the 2nd rib the inferior angle reaches as low as the 7th interspace or 8th rib

The 12th rib cannot always be felt and so it is not wise to count the ribs from below upwards These are best counted downwards from the second costal cartilage This cartilage articulates with the sternum at the extremities of the angle of Louis a transverse bony ridge at the junction of the body and the manubrium which is easily felt beneath the skin This angle is a useful landmark for in addition it indicates the level at which the trachea bifurcates the upper point at which the anterior borders of the lung meet in the mid line and the level of the upper border of the cardiac auricles

Anatomy of the bronchi —The two main bronchi each give off four main branches on the right one to the upper lobe one to the middle lobe one to the dorsal lobe (the upper and posterior part of the lower lobe) and one to the remainder of the lower lobe and on the left one to the upper lobe proper one to the lingular process of the upper lobe (which represents the middle lobe on the left side) and a dorsal and lower lobe bronchus as on the right side These main bronchi then divide into segmental bronchi which supply individual segments of lung It is important to have a working knowledge of these segments because it is often possible from the signs and X ray appearance present to determine which segment and which segmental bronchus is affected by disease The accompanying diagrams (Figs 50–2) give a simplified scheme of present knowledge of the anatomy of the segmental bronchi and indicate the respiratory districts supplied by them

II INSPECTION

A FORM OF THE CHEST

This depends partly on the curvature and obliquity of the ribs partly on the curves of the spinal column The curvature of the sternum results from the relations of these factors

The **ideal healthy chest** conforms to the following description It is bilaterally symmetrical its contours are smooth it has no deep hollows and at most shows only a slight recession below the clavicles In cross section it is an ellipse, broader from side to side than from front to back in the proportion of about 7 to 5 its general shape is ellipsoidal with the longest axis vertical In children the cross section is more nearly circular In practice it is rare to find a chest which is perfectly symmetrical

To inspect the chest the examiner should first look from the front then from the side thereafter from the back and finally *over the shoulders from behind and above to see the profile of a* horizontal section of the thorax The last method is useful in detecting lack of symmetry or unequal expansion on the two sides The set of the neck on the chest and the epigastrium should be inspected at the same time as the thorax In examining the chest from behind note whether kyphosis and scoliosis are present

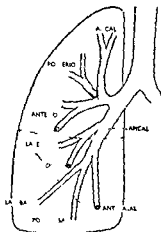
The shape of the chest varies with the build of the individual often being short broad and deep in the thick set and long flat

The Right Lu g

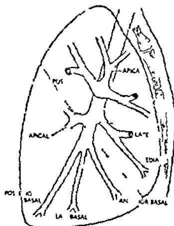
Upper lobe
- Ap cal bronch s and segment
- Post r o bronchu and segm nt
- Ante or b onchus and segme t

M ddle lobe
- Late l b onchus a d egment
- M d al bronchus and segm nt

Lower lobe
- Apical bro chu a d gment
- Med al basal bron.hus nd s gme t (bronchus not sh wn on d agr m)
- Ant rior b sal bronchus and egm nt
- Lat ral basal b on hus and *s gm nt*
- Post r o bas l b o ch s and *s gment*



R ght lung—A t r o a p ct



R ght lung—Lateral spect

Fig 50 —Right lung showing diagram of segmental bronchi

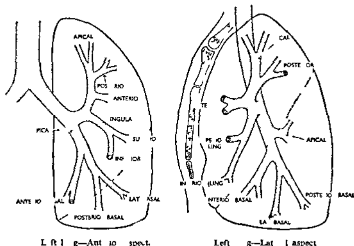
and narrow in the tall and spare There is also wide variation in the thickness of the muscles and the slope of the shoulders in healthy people

Estimates of the significance of variations in the shape of the chest must therefore take into account the build of the individual In the past rickets has been a common cause of chest deformity the combination of softness of the bones and obstruction to respiration due to adenoids and chronic or recurrent upper res

Th L f Lu g

Uppe l be
- Upp d n b o ch
  - p cal b h d segment
  - post b nch a d segme t
  - t nor b h d segm t
- Lung l (low d o) bro h s
  - p b h d s gm t
  - inf b o h d gme t

Low l be
- Ap l b o h d segm t
- A t b sal bron h d gm t
- Lat l b sal b h s d segm nt
- P t ri basal b on h d gm t



L ft l g—Ant io spect. Left g—Lat l aspect

Fig 51 —Left lung showing diagram of segmental bronchi

piratory tract infection leading to the deformities known as pigeon chest and Harrison s sulcus

The pigeon breast —Here the sternum becomes unduly prominent and projects beyond the plane of the front of the abdomen so that there is a sharp angle at its lower end At the same time the cross section of the chest ceases to be elliptical and approaches a triangular form the angles being situated at the sternum in front and at the costal angles behind

Harrison s sulcus —This is a transverse constriction which beginning at

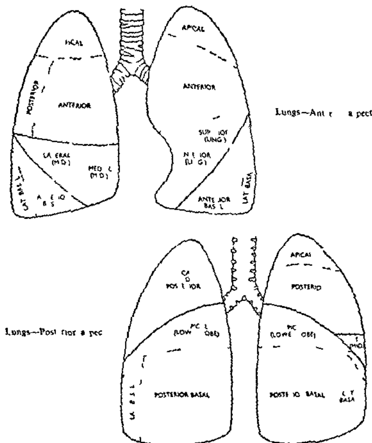
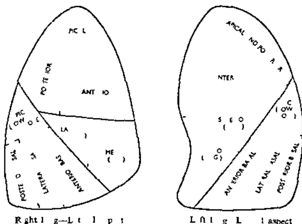


Fig 52 —Diagram of respiratory di tricts upsplied by segmental bronchi

(F gs 50–2 a e eprod c d ith th Alod permis i of D Robert Coop a 1 M s s E & S Li ing to e f om Kee s and Bigd "P lm na y Tuberc lo i)

the level of the xiphisternum passes outwards and slightly downwards It seldom reaches as far as the midaxillary line

The presence of these deformities indicates that the patient has



Right lung—Lateral aspect Left lung—Lateral aspect
Fig. 52 —(*cont*)

suffered from rickets and they are becoming rarer as the nutrition of young children improves

The commonest causes of chest deformity not due to disease of the thoracic viscera are **scoliosis** and **kyphosis**

Scoliosis because of curvature of the spine and rotation of the vertebræ leads to undue prominence of the front of the chest on that side with flattening on the other side Because of the rotation of the vertebræ the actual curvature is greater than appears from inspection of the posterior vertebral spines The most obvious signs of scoliosis are usually an undue prominence of the scapula on the side of the convexity and undue prominence of the hip on the opposite side Rarely scoliosis may be induced by fibrosis of the lung in a young subject

Kyphosis leads to shortening of the chest and undue prominence of the sternum and when there is a gibbus or angulation of the spine as from healed tuberculous caries the deformity may be extreme A kyphotic chest may simulate an emphysematous one

In the **barrel shaped chest** the ribs are less obliquely set than usual the antero posterior diameter of the chest is increased the spine becomes unduly concave forwards and the sternum is more arched than under normal conditions while the angle of Louis becomes more prominent The chest thus becomes fixed in what

is normally the position of full inspiration Since further inspiration can only be effected by movements of the chest as a whole the accessory muscles of respiration have to take the place of the intercostals thereby becoming abnormally conspicuous and respiration becomes mostly abdominal The barrel shaped chest is usually associated with chronic hypertrophic emphysema

Unilateral enlargement is found very rarely in large pleural effusions Gross cardiomegaly in children may produce local prominence of the chest wall

Diminution of volume of one side results from shrinkage of the lung This may be caused by fibrosis or it may be the result of adhesions Collapse of a lung from obstruction of the bronchi may produce a similar result Before connecting these changes with disease of the lungs or pleura the observer must ascertain that no scoliosis exists

Local bulging — In emphysema the apices may produce an unusual fullness above the clavicles and in pleural effusion especially if purulent the interspaces which lie in the area of effusion may bulge considerably though this is a rare finding

Tumours of various kinds abscesses of the chest wall and great enlargement of the heart or pericardial effusions in children may rarely be the cause of localized bulging

Local shrinking —In phthisis one or both apices are often contracted and a hollowing is produced above or below the clavicles

To detect either bulging or flattening (as well as diminished expansion) it is important to look tangentially along the chest either from above the shoulders or upwards from below Both in phthisis and in other wasting diseases the interspaces are very sunken and the ribs prominent in consequence of the malnutrition of the muscles and subcutaneous tissues

A funnel shaped depression is sometimes found in the lower part of the middle line of the thorax in front Sometimes it is congenital or it may be developed in infancy with or without any obstruction to respiration being present It extends in some cases as high as to the 3rd rib

B MOVEMENTS OF THE CHEST

The movements of the chest during respiration also demand attention and the rate of movement its rhythm its type and its amount, must be noted

1 The rate for an adult in health is about 18 or 20 respirations per minute but there is a wide margin on either side of these figures Increased rate may result from exertion nervous excitement fever or anoxæmia which may be due primarily to cardiac pulmonary bronchial or laryngeal causes to alteration in the oxygen-carrying power of the blood or to interference with the normal reflex control of respiration (the Hering Breuer reflex) by structural changes in the lungs It may also arise from the association of pain with breathing as in pleurisy and peritonitis when the breathing becomes shallow and must therefore be more frequent to make up for the slighter expansion

The ratio between respiration and the pulse in health is about 1 to 4 in severe pneumonia respiration may occur almost as frequently as the pulse in certain cases of narcotic poisoning the ratio may become 1 to 6 or 7

2 The rhythm varies very considerably even in health and if the act is performed consciously it may become very irregular Study it therefore when the patient is off his guard as only then can accurate observations be made Either inspiration or expiration may be unduly prolonged the former being commonly associated with laryngeal or tracheal the latter with bronchial or pulmonary diseases A peculiar type where successive respirations gradually get deeper and deeper till a maximum is attained and then fall off again until a pause of complete apnœa occurs to be followed by another wave of gradually deepening and then diminishing respiration, is known as Cheyne Stokes breathing The pause may last for fully half a minute though it is often shorter and the whole cycle is usually completed in less than two minutes It is most conspicuous when the patient who exhibits it is asleep or unconscious but may be overlooked if the patient is awake and particularly if he is talking Cheyne Stokes breathing occurs most commonly in cardiac and renal failure severe pneumonia, increased intra-cranial pressure and narcotic drug poisoning Another rare

form of respiration irregular in rhythm and in depth with irregular pauses and occasional sighs but not periodic is sometimes observed in cases of meningitis and is known as the Biot type of respiration

3 Type —Breathing may be more evidently performed by the upper part of the thorax thus is known as the thoracic type of respiration It is found to a certain degree in women and in hysterical or anxious subjects but in its full development is either associated with paralysis of the diaphragm or else is a result of increased abdominal pressure as in ascites

In men and young children the diaphragm and abdominal muscles play the most important part in respiration and in cases where some painful condition exists in the thorax the breathing may be wholly abdominal in type

In paralysis of the diaphragm there is in addition increased horizontal expansion of the lower part of the chest and widening of the subcostal angle with recession in place of the normal expansion of the epigastrium and hypochondria in inspiration In paralysis of the intercostal muscles there is a diminution in the size and expansion of the chest, with narrowing of the subcostal angle and increased diaphragmatic movements

The presence of pain or breathlessness should always be asked for and its exact nature noted (*see* Chap II p 7)

4 Regarding movement during respiration the points to be noted are its amount whether it is expansive in character and whether it is similar or different on the two sides and over corresponding areas Amount of movement and expansion are by no means interchangeable terms in emphysema the chest may move considerably but there is little expansion

Diminished or absent expansion may be due to pleurisy with effusion pneumothorax consolidation collapse fibrosis or the presence of a neoplasm It is also found in tuberculosis and lobar pneumonia the former especially at the apices the latter at the apex or base according to the situation of the disease

Sometimes one part of the chest wall lags behind the rest during inspiration. Any such lagging is suggestive of disease The existence of any indrawing of the intercostal spaces during inspiration or of any bulging during expiration must be noted Both may occur physiologically when the conditions are present over the

whole chest and are not very conspicuous they may result from pathological conditions Examples of inspiratory indrawing are found in obstruction of the larynx (general) or in blocking of some of the smaller bronchi (local) One of the best instances of localized expiratory bulging is seen at the apices of the lungs in advanced emphysema and this may be associated with indrawing of the lower ribs in inspiration

III PALPATION

Palpation takes note of the form and movements of the thorax and the position of the apex beat and trachea of vibrations or tremors which are communicated to the hand and of the behaviour under pressure of any pain of which the patient complains Under the first head inspection is supplemented under the second one may learn something of the accompaniments—e g friction or rhonchi—which interrupt the smoothness of the respiratory move ments and also of vocal fremitus which serves to indicate the condition of the conducting media. The third enables one to detect the cause of many thoracic pains

Before making a systematic examination it is well to lay the hand on any part of the chest which presents an obvious swelling or where the patient complains of pain Look at the face rather than the part under examination so as to avoid causing unnecessary pain Pain may be due to recent injury to the chest wall or to inflammatory conditions to intercostal muscular pain where as a rule specially painful spots can be discovered on pressure to herpes zoster before the appearance of the eruption or to pleurisy In the case of pleurisy pressure may considerably increase the pain Pain may also be due to cardiac causes such as coronary thrombosis and pericarditis At the same time the nature of any swelling should be investigated Fluctuation occurs when an abscess has formed in the chest wall Such an abscess may be due to disease of the bones or soft parts forming the thoracic wall or rarely to pus which has broken through from the pleural cavity (*empyema necessitatis*) In the latter case the pus may be driven back by gentle pressure to reappear when the patient coughs

The observer should then measure the expansion of the chest with a tape measure In a well formed adult male the girth of the chest at the level of the nipples should be 34 in at the end of

expiration and should measure at least 2 in more when a deep inspiration has been taken Height age and build modify these measurements different races vary considerably in chest girth

The positions of apex beat and trachea should then be determined Feel for the trachea in the suprasternal notch and decide whether it is placed centrally or deviated to one or other side by its relation to the suprasternal notch and the insertion of the sterno mastoids Displacement of the apex beat alone may be due to scoliosis the commoner form with its convexity to the right causing a displacement of the apex beat to the left and vice versa to funnel depression of the sternum or to enlargement of the left ventricle particularly in aortic incompetence and hypertension In the absence of these conditions a significant displacement of the apex beat or trachea or of both together suggests that the position of the mediastinum has been altered by disease of the lungs or pleura The main conditions which push the mediastinum away from the affected side are pleurisy with effusion and pneumothorax and the main conditions which draw it towards the affected side are fibrosis of the lung in tuberculosis or after broncho pneumonia and collapse of one or more lobes

The **nature of the respiratory movements** must next be studied It is important to make certain that the two sides of the chest move to approximately the same extent This is done by fixing the finger tips of either hand at the patient s sides and making the tips of the thumbs just meet in the middle line in front of the chest The patient is directed to take a full inspiration when the distance of departure of the thumbs from the middle line indicates the extent of expansion of either half of the chest The causes of diminished expansion have been mentioned under inspection

Sometimes one half of the thorax lags behind the other this is detected by the hands no longer moving synchronously

The movements at the apices may be similarly observed In this case the physician stands behind the patient and fixing his thumbs on the vertebræ lets his fingers lie over the right and left lung apices reaching towards the clavicles whilst the patient breathes deeply

Vibrations may be detected by palpation *For this purpose the palm or ulnar border of the hand should be applied flat on the chest* and since the sensitiveness of the two hands is often unequal *the same one should be employed on both sides* In addition to the

vibrations already referred to in Chap IV (p 83) fremitus may be due to pleural friction or to catarrhal changes in the mucosa of the bronchi leading to local constrictions that is to palpable rhonchi After the presence or absence of these forms of fremitus has been determined the observer should study the **vocal fremitus** or vibrations which the voice communicates to the chest wall These are conducted from the larynx by the trachea and bronchi to the smaller tubes within the lungs and thence through the lung tissues to the surface Anything which affects the conducting power of the air passages or lung tissue or the interposition of additional materials through which the vibration must pass to reach the palpating hand will affect the intensity of the fremitus To test the vocal fremitus the patient is told to repeat one one one or ninety nine in a clear voice The hand placed on the thorax detects distinct vibration whilst this is done and it must be determined whether the vibrations in corresponding areas on the two sides of the chest are approximately equal in intensity—not, however forgetting that where the heart encroaches on the left lung the fremitus is necessarily much diminished—and also whether they correspond to what former experience has led the observer to recognize as normal for the region under examination for a similar chest and like pitch and loudness of voice **Vocal fremitus is increased** when the lung is consolidated as in lobar pneumonia or contains a large cavity near the surface **Vocal fremitus is diminished** when the corresponding bronchi are obstructed or totally absent when the lung is separated from the chest wall by pleural effusion or hydrothorax The cause in this case is not that fluid is a bad conductor of sound or of vibration—the reverse is the case—but that the relaxed lung itself fails to convey the vocal fremitus and so the vibrations never reach the fluid In young persons and in female subjects the vocal resonance is different both in character and intensity from that which occurs in male adults The resistance of the chest to compression is best estimated by placing the hand over the sternum whilst the patient is lying down and attempting to press it backwards towards the vertebral column The rigidity increases with advancing age and also in certain diseases (e g in tuberculosis and in emphysema)

IV PERCUSSION

Methods of percussion —When percussion was first introduced the tap was delivered directly on the patient s skin without the interposition of any substance over the point struck This method known as *direct percussion* is now seldom used except on the clavicles which in examination of the lungs are lightly tapped by the observer s finger tip

The ordinary or indirect, method of percussion is conducted in the following manner The middle finger of the left hand is placed *firmly* on the part which is to be percussed and is adapted to any inequalities of surface so that no air space is interposed between it and the skin The back of its middle phalanx is then struck with the tip of the middle finger of the right hand The stroke should be delivered from the wrist and finger joints not from the elbow and the percussing finger should be so bent that when the blow is delivered its terminal phalanx is at right angles to the metacarpal bones and strikes the pleximeter finger per pendicularly As soon as the blow has been given the striking finger must be raised lest it should impair the vibrations it has excited just as the hammers of a piano fall back from the wires as soon as these have been struck The blow should be no heavier than is necessary to elicit the resonance of the part being examined and the wrist joint must move loosely Good percussion is a knack which requires much practice

Three cardinal rules should always be remembered when per cussion is being carried out The first is that in defining the boundaries between contiguous organs the percussion should invariably be performed from the resonant towards the less reso nant The second is that the longer axis of the pleximeter finger should be parallel to the edge of the organ whose delimitation is being attempted and the line of percussion should be at right angles to that edge The third is that the pleximeter finger must be kept in firm contact with the chest wall

It is seldom necessary to deliver more than two or three strokes at any one situation repeated blows cause much discomfort to a sensitive patient. The points to be noted on percussion are the *volume* and *pitch* of the resonance elicited and the sense of *resistance* experienced by the finger

The character of the sound produced varies quantitatively and

qualitatively the quantitative variations depending on the force of the blow delivered and on the capacity of the part struck to resound to the blow The quality of the sound depends on the particular vibrations which are elicited and on the selective reinforcement of some of them by the resonance of the chest wall and of the organs involved

When the air in a cavity of sufficient size and appropriate shape is set into vibrations which are not modified by excessive tension of the containing walls of the space the sound heard has a tympanitic character such a note is heard on percussion over an air-containing viscus such as the stomach but when the cavity is sub-divided into a number of small loculi by numerous septa more or less tense a characteristic resonance no longer tympanitic is produced. Such conditions prevail in the healthy lung, and the observer must learn by practice to recognize its distinctive quality In general terms this pulmonary resonance is low in pitch and clear in character

In percussion over the lung we endeavour to ascertain three sets of facts first, the limits of the lung resonance second, the state of the lungs in regard to the quantity of air contained in their various parts and third whether they are unusually remote from the surface of the chest, the separation being due to thickened pleura or to fluid or gas in the pleural cavity

Beginning in front, the examiner should tap lightly on the most prominent point of each clavicle—being sure that the points examined correspond exactly with each other—and should observe the quality of the sound, and particularly whether the effects on the two sides are identical Thereafter the other corresponding areas on either side should be carefully compared many points being systematically percussed in each area The presence of the heart will interfere in parts of the left side with the development of a sound resembling that from the corresponding point on the right.

When the front has been fully examined the observer should percuss in both axillary and infra axillary regions—the patient holding his hands joined above his head lastly the various areas posteriorly should be worked out the patient, if able to sit up being instructed to fold his arms and bend slightly forwards

It is essential in all parts of the examination that the patient's attitude is a comfortable one and that his arms and shoulders are placed symmetrically whether he is sitting up or lying down.

His muscles must be relaxed percussion over tense muscles tending to produce a deceptively dull and short lived note The head must not be inclined to either side When in the case of a very ill patient it is only possible to examine the back by rolling him on to his side the comparison of the two sides by percussion is seriously interfered with and only gross differences in the note on the two sides are significant *If possible the patient should be examined lying first on one side and then on the other Should the patient s chest be asymmetrical from scoliosis or other cause equal resonance on the two sides is not to be expected* and again only gross differences between the two sides are significant

The observer should have two objects in mind first to make a comparison of the percussion note in comparable areas on the two sides and second to map out the limits of lung resonance particularly at the apices the bases and the area of cardiac dullness

The normal degree of resonance varies from individual to individual and in different parts of the chest in the same individual being most resonant below the clavicles and scapulæ where the muscles are relatively thin and least resonant over the scapulæ Long practice only will enable the student to estimate normal resonance and what degree of firmness of percussion is necessary to elicit it

The resonant areas at the front and the back of the chest are connected by a band of resonance which passes over the shoulder between the dullness due on the inner side to the structures of the neck and on the outer side to those of the shoulder joint This band known as Kronig s isthmus is normally some 4 to 6 cm wide Its narrowest point is at the apex of the shoulder from which *it widens out to merge with the infraclavicular resonance anteriorly* and with that below the spine of the scapula posteriorly Narrowing or obliteration of this band is a sign of disease usually tuberculosis at the apex

The lower limits of lung resonance should be determined by percussing from above downwards *The lower border of the right lung lies over the liver* and *is thin therefore its exact situation is best made out by light percussion Posteriorly* however the muffling due to the thick muscles and fat of the back makes it necessary to percuss more firmly When the patient is obese very heavy percussion with several fingers may be necessary in order to penetrate the parietes and bring the lung tissue within influence

of the blow In quiet respiration and on light percussion the lower border is found to lie in the mammary line at the 6th rib in the midaxillary line at the 8th rib in the scapular line at the 10th rib and nearer the vertebral column as low as the 10th space On heavier percussion some loss of resonance due to the under lying liver and diaphragm is found at higher levels and in the mammary line can be detected from the 4th interspace downwards

On the left side the lower border overlaps the stomach and so the transition is not from lung resonance to dullness but to tym panitic stomach resonance Posteriorly however the splenic dull ness and the dullness of the various solid structures which lie below the lung near the spine are interposed so that the conditions resemble those found on the right The position of the lower border corresponds pretty closely with that on the right side it may however be found a trifle farther down

Percussion of the area of cardiac dullness has already been discussed (p 83)

The limits described are exceeded in very deep inspiration, and in emphysema where the volume of the air-containing lung is increased and where the cardiac dullness may be much diminished or absent In pneumothorax the lower border of resonance is often considerably below the limits assigned and the character of the sound is different (*see* below)

Since in health the borders of the lungs have a considerable range of movement during deep respiration whilst in the presence of disease the range is often much restricted it may be useful to percuss the lower borders of the lungs during both expiration and full inspiration A unilateral diminution or absence of movement at the base may be an early sign of fixation of the diaphragm It is found for instance in subphrenic abscess and in liver abscess encroaching on the diaphragm

In disease the resonance may be affected (1) quantitatively and (2) qualitatively

1 **Quantitative** —**Resonance is increased** when the pleural cavity contains air and the lung is more or less collapsed towards the hilum The note varies from one that is hyper resonant to one that is distinctly tympanitic according to the amount of air in the pleural cavity

A characteristic form of high pitched tympanitic resonance the

bruit d airain (*airain* = brass) bell sound or coin sound may also sometimes be heard in pneumothorax by percussion over the front of the chest with a couple of coins—one being used as a plexor and the other as a pleximeter—whilst the observer listens with the stethoscope at the back of the patient In very marked cases the sound is soft and musical and has been compared with the stroke of the hammer on an anvil when heard a long way off Failure to elicit the bruit d airain does *not* mean that a pneumothorax is not present Large cavities within the range of the percussion stroke may likewise produce a hyper resonant or tympanitic note under certain conditions but many cavities do not do so

Resonance is increased to some degree in most cases of emphysema but the character and pitch of the note obtained may be so altered by the increased rigidity of the chest wall, as almost to suggest dullness in some cases Resonance may also be increased on percussion over the lung above the level of the pleural effusion or in the upper portion of a lung whose lower lobe is affected by pneumonic consolidation This phenomenon is known as skodaic resonance

Resonance is diminished when the pleura is thickened when the underlying lung is more solid than usual for any reason and when the pleural cavity contains fluid Thus there may be slight impairment of resonance usually at one or other apex in a case of pulmonary tuberculosis when by reason of varying degrees of infiltration and fibrosis the lung tissue is less well aerated than normal Considerable impairment may be found over areas of lung affected by a pulmonary neoplasm a lung abscess fibrosis or collapse In lobar pneumonia, percussion over a completely consolidated lobe produces a definitely dull note whilst absolute dullness along with a peculiar sensation of resistance in the percussing finger so-called stony dullness is the characteristic finding over a pleural effusion of any size In heart failure impaired resonance or dullness may be found at the bases of both lungs indicating œdema of the bases or bilateral effusions

2 *Qualitative* —Two peculiar sounds which are produced by percussion in pathological conditions remain to be noted

Cracked pot sound —This is due to a sudden expulsion of air through a constricted orifice It occurs in cases where percussion is practised over

a large cavity which communicates with a bronchus of moderate size and is most distinct when the mouth is opened It is a rare finding If healthy children are percussed while they are crying a similar cracked pot sound is often produced

Amphoric resonance —This phenomenon is due to the selective reinforcement of certain vibrations by a large cavity by this means the overtones are accentuated and die out more slowly It is a low pitched hollow sound which may occasionally be heard on percussing over a pneumothorax or large cavity

Myotatic irritability —In certain conditions the muscles on the front of the thorax are unduly irritable and a light tap over the sternum produces contractions at some distance off in the pectoral muscles This phenomenon occurs in any wasting disease and often in tuberculosis and is known as myotatic irritability or myoidema

V AUSCULTATION

Three observations must be made at each point auscultated first the character of the breath sounds second the character of the vocal resonance and third the presence or absence of other sounds

For good ausculation a patient in bed should lie on his back and be completely relaxed To examine the back, the patient should sit up but if he is unable to do this he should be rolled round first to one side then to the other In serious cases injury may be done to a patient by prolonged examination Take care that the chest piece is accurately applied that it is not allowed to move on the surface of the skin and that no undue pressure is exerted The patient should breathe with his mouth open regularly and fairly deeply but not noisily It is quite useless to attempt auscultation of a patient who is shivering

A CHARACTER OF RESPIRATORY SOUNDS

There are two typical varieties of breath sounds both of which are audible in health at certain parts of the chest and these must be carefully studied The first is known as vesicular breathing the second as bronchial Vesicular breath sounds are produced by the passage of air in and out of normal lung tissue and are heard all over the chest under normal conditions Bronchial breath sounds are produced by the passage of air through the trachea and

large bronchi Under normal conditions they can be heard by listening over the trachea but are not heard over normal lung tissue (except where they may modify the sounds heard over normal tissue situated near the trachea and large bronchi as will be mentioned later) In disease however conditions may become favourable to the conduction of these sounds from the bronchi to the chest wall as for instance when a whole lobe is consolidated by pneumonia Under these circumstances no air enters or leaves the alveoli and no vesicular breath sounds can be heard Provided however that the bronchi are patent so that bronchial sounds are produced and conducted through them and provided that sufficient lung is consolidated to convey these sounds to the chest wall *bronchial breathing will be heard over the area affected by these changes*

In **vesicular breathing** which can be heard typically in the axillary and infrascapular regions of a healthy individual the following facts pertain —

The inspiratory sound is fairly intense and is audible during the whole of the act The pitch is low and the quality is characteristic being somewhat rustling The expiratory sound follows that of inspiration without a distinct pause—unless as often happens the patient holds his breath for a second at the end of inspiration It only remains audible during the earlier part of the expiratory phase and under normal conditions the inspiratory sound is heard for at least twice as long as the expiratory

To learn to recognize **bronchial** breathing the student should listen over the trachea though he must not expect to hear so intense a type of bronchial respiration when he subsequently examines a diseased lung The inspiratory sound is moderately intense It becomes inaudible shortly before the end of inspiration Its quality is blowing or hollow with a guttural or aspirate intonation The expiratory sound is generally more intense than the inspiratory the pitch is often higher the duration extends through the greater part of expiration being as long as or even longer than the inspiratory sound which often follows without any pause In quality it exactly resembles the inspiratory sound being aspirate or guttural in character Although vesicular breathing is characteristically rustling and bronchial aspirate or guttural the essential difference between the two is the difference in rhythm that has been described

1 The principal variations which can be detected in vesicular breathing are as follows —

i **Puerile** —The sounds are harsher than in the adult but have a similar duration

ii **Jerky interrupted or cog wheel inspiration** —Here the sound is not continuous but occurs in waves or sharp jerks Although sometimes present in early tuberculosis it may result simply from nervousness and so carries little weight as a physical sign

iii **The breath sounds may be reduced in intensity or absent** and this may affect the whole chest or be confined to one or more areas In persons breathing quietly in health the breath sounds may be almost inaudible This has no pathological significance and the sounds become audible if the subject is asked to breathe more deeply

Local diminution or absence of the breath sounds in one or more areas may however be an important sign of disease It is found in the presence of greatly thickened pleura pleural effusions and pneumothorax It is also found in any condition causing a diminished air entry to the underlying lung when the conditions necessary for the appearance of bronchial breath sounds are not present It may be found for example in pulmonary tuberculosis —usually at one apex in lobar pneumonia before consolidation is established—usually at one base in heart failure usually at both bases or over areas of lung affected by neoplasm fibrosis or collapse from any cause

Over a *pleural effusion* breath sounds of any kind are absent as a rule probably because though fluid itself is a good conductor of sound a combination of partly collapsed lung and fluid does not form a sufficiently uniform conducting medium Occasionally it happens that when a considerable quantity of fluid has accumulated the breath sounds instead of disappearing become loud and possess a marked bronchial character In such cases the vocal resonance also is loud but is usually more or less ægophonic This exceptional state is most commonly observed posteriorly over the lower lobe of the lung in children and may be due to a complete collapse of part of the lung enabling the vibrations which are present in a bronchus to be transmitted to the fluid with less loss

of intensity than if they had first required to pass through air containing lung and thus providing a more uniform sound conducting medium

Some prolongation of the expiratory sound is characteristic of asthma and emphysema and this must not be mistaken for bronchial breathing It is due to the fact that in these diseases the act of expiration is itself performed more slowly than in health

2 **Bronchial breathing** is subdivisible into three varieties according as the bronchial respiratory sound is conveyed to the ear through consolidated lung from the larger medium or small air passages each of which by reinforcing certain elements gives it a distinctive pitch and character

In the first case we have **low pitched bronchial breathing** in the second case the pitch is **medium** in the last it is **high** Low pitched bronchial breathing is heard over moderately large cavities in the lungs and is hence sometimes called *cavernous* high pitched bronchial breathing is heard when consolidation has occurred round the smaller tubes as in pneumonia where the most perfect examples of bronchial breathing may often be found Here the character is aspirate rather than guttural This variety is often known as *tubular breathing*

A fourth and special variety of bronchial breathing is known as **amphoric respiration** It resembles the sound produced by blowing across the mouth of a bottle and consists of one or more low pitched fundamental tones and a number of high pitched overtones It is characteristic of a direct communication between the bronchus and either a considerable cavity with fairly smooth walls or a pneumothorax The latter condition yields the best examples

When breath sounds in a superficial bronchus can be heard through normal lung the sound of the breathing combines both vesicular and bronchial elements one or other type predominating according to the exact relations in each case This variety of breath sound is known as **broncho-vesicular** and it is usually the expiratory sound which has more of a bronchial character It occurs in health near the roots of the lungs behind in the upper portions near the middle line in front and especially at the right apex for a few centimetres below the clavicle in front and above the level of the spine of the scapula near the mid line behind In the same areas vocal fremitus and resonance may be increased and some degree of whispering pectoriloquy may be heard These

indings which sometimes lead to a mistaken diagnosis of disease t the right apex are due to the fact that the trachea lies in imme iate contact with the apex of the lung on the right side whereas t is separated from it on the left by the aorta the internal carotid rtery and the œsophagus

The breath sounds must be auscultated in the various regions hat have already been examined by percussion their character n each noted and similar regions on the two sides of the chest ompared care being taken that the points examined correspond ccurately to one another

If the student understands how vesicular and bronchial breath ounds are produced he should have no difficulty in explaining the ypical findings in disease Vesicular breath sounds may be present but reduced in intensity in any condition in which the entry of air o that part of the lung is diminished as for instance in fibro aseous tuberculosis where some alveoli are affected and others not Breath sounds of any kind may be diminished or absent where hickened pleura pleural effusion or pneumothorax interferes with or prevents the conduction of these sounds to the chest wall They may also be absent in any condition such as collapse or fibrosis n which no air enters or leaves alveoli but at the same time the onditions necessary for the conduction of bronchial breath sounds o the chest wall are not fulfilled Finally bronchial breath ounds of various kinds may be heard whenever patent bronchi are connected to the chest wall by a sufficiently uniform sound onducting medium This occurs classically in the consolidation of obar pneumonia occasionally in the presence of a very large bron hial carcinoma and in conditions previously mentioned

B VOCAL RESONANCE

The second series of observations is directed to the intensity and character of the vocal resonance It varies in intensity even in health on the two sides and over different areas of the lung being lightly louder on the right side and more intense the nearer the tethoscope is to the larger bronchi When the patient repeats he words one one one or ninety nine the ear receives rom the chest no distinct impression of the syllables pronounced but only a buzzing sound whose intensity depends on the loudness and depth of the patient s voice and on the conductivity of his lungs

Each point examined on one side of the chest should be ^*

compared with the corresponding point on the other side Vocal resonance of normal intensity generally conveys the impression of being produced just at the chest piece of the stethoscope If it seems to be nearer the ear than this the resonance is increased When it appears to be near the ear piece of the stethoscope the increase is marked and the condition is often described as **bronchophony**

If the words become clear and seem to be spoken right into the auscultator s ear it will generally be found that whispered words are distinctly heard This condition is called **whispering pectoriloquy** Increased resonance occurs when the lung substance conducts the sound waves set up by the voice more clearly than usual from the bronchi Consolidation is the commonest cause of increased lung-conductivity Bronchophony and whispering pectoriloquy occur when a moderately large bronchus is surrounded by a layer of solid lung reaching to the chest wall as in lobar pneumonia Whispering pectoriloquy is also fairly characteristic of a cavity of some size communicating with a bronchus and may be heard above the level of a pleural effusion In some cases a certain degree of pectoriloquy is heard in health in the proximity of the trachea and large bronchi and particularly at the right apex

For reasons already explained vocal resonance is either entirely abolished or much diminished where a layer of fluid separates the lung from the chest wall (except when loud bronchial breathing is heard—*see* p 199) and in pneumothorax It is also diminished *in cases of thickened pleura and of emphysema*

In certain conditions the quality of the vocal resonance undergoes modification Above the level of a pleural effusion or in some cases over an area of consolidation a nasal or bleating character may be imparted to the voice This bleating tone is observed much more frequently at the back near the lower angle of the scapula or between that point and the axillary line than it is over other regions of the thorax It is known as **ægophony** and it is probable that the peculiar quality of the voice is due to the fundamental tone being intercepted to a much greater degree than the overtones

C ADDED SOUNDS

These may arise in the lung or in the pleura Sounds resembling pleural friction may be produced by movement of the stethoscope

on the patient s skin or of the observer s hands or clothes against the stethoscope Sounds arising in the patient s muscles may resemble adventitious sounds and in particular the shivering of a cold patient makes any attempt at auscultation useless The application of the stethoscope to hairy skin may produce sounds indistinguishable from crepitations These can be suppressed by moistening the skin or removed by shaving it Sounds resembling coarse crepitations may also be heard over a broken rib

Dry sounds known also as **rhonchi** and produced in the air passages are continuous snoring sounds and are due to partial obstruction of their lumen either by swelling of the mucosa or by the presence of tough secretion The mechanism of their production is comparable with that of cardiac murmurs They vary in pitch the variations being largely due to the size of the tubes where they take origin The smaller tubes produce high pitched or **sibilant rhonchi** which are most abundant during the latter part of inspiration the medium sized tubes yield medium pitched rhonchi and the larger bronchi produce the deep toned or **sonorous rhonchi** which are heard early in inspiration and may be almost continuous Dry sounds are characteristic of bronchitis but are also found in other diseases such as cases of tuberculosis when the bronchial tubes get plugged Rhonchi are often heard throughout expiration especially in asthma and in bronchitis with bronchospasm

Moist sounds (crepitations) are discontinuous crackling sounds produced either in the alveoli or in the bronchioles and bronchi They sound like the bursting of small air bubbles and indicate the presence of fluid secretions in the air-cells or tubes They are classified as fine medium and coarse The term crepitation is sometimes restricted to the first variety the others being called moist rales

Fine crepitations are probably caused by the opening up of collapsed alveoli whose walls have been agglutinated by the exudation of a little fluid secretion The separation of the walls is accompanied by a clicking sound and when this condition occurs in a number of alveoli the combined effect is to produce a sound of fine crepitation which can be imitated by rolling a few hairs between the finger and thumb in front of the ear It occurs only near the end of inspiration, and indicates the presence of exudation in the alveoli of the affected part of the lung Fine crepitations are

characteristically present during the first stage of pneumonia at the apices in tuberculosis and at the bases in heart failure

Medium crepitations occur chiefly in the smaller bronchi and are audible at the end of inspiration and the beginning of expiration They are caused by the air bubbling through fluid secretion which has been poured out into the lumen of the bronchi

Coarse bubbling crepitations occur in the larger divisions of the bronchi and may be heard at almost any phase of respiration they may be quite continuous Medium and coarse crepitations are heard particularly in bronchitis and may also originate in pulmonary cavities

When a patient has been lying quiet for a time without breathing deeply a few crepitations may be heard in health particularly at the lower borders of the lungs These are abolished if the patient is asked to cough and are of no significance In other cases crepitations are intensified after a cough or may only then make their appearance After auscultation has been carried out in the normal manner the patient should be asked repeatedly to give a small cough and follow it by taking a breath while the observer listens for crepitations in different areas These crepitations brought out by coughing are known as **post tussive crepitations** They are an important sign of tuberculous infiltration and may also be heard over cavities

The most common adventitious sound arising in the *pleural cavity* is a **friction sound or pleural rub** characteristic of pleurisy at the stage when exudation is not abundant enough to separate the inflamed and roughened surfaces It has a creaking or rubbing character often quite characteristic but sometimes rather hard to distinguish from crepitation The friction sound may be fine medium or coarse In some instances it is palpable but since coarse crepitations may be so too this does not distinguish them The chief features of difference are that friction sounds occur during that part of inspiration when the roughened surfaces are rubbing against each other to reappear at a corresponding period of expiration They are moreover unchanged after the patient has coughed whilst crepitations may alter under these conditions because of changes in the disposition of the secretion which causes them Friction is sometimes more localized than crepitations Sometimes friction is intensified by pressing hard with the stethoscope This causes the roughened surfaces to rub against each

other more firmly Pressure does not affect the intensity of crepitations The situation of the doubtful sound the presence of pain or some point in the history of the case may help in arriving at the diagnosis

The presence of one form of accompaniment does not exclude the others Any two or three may be found coexisting in one case When pleuritic friction is developed along the anterior edge of the left lung and especially when that part of it which is in relation to the apical segment of the heart is affected the friction sounds often assume the rhythm of the heartbeat rather than that of the respiratory movements Hence the sound is liable to be mistaken for pericardial friction To distinguish between this so-called *pleuro pericardial friction* and that of true pericarditis ask the patient to hold his breath. This usually abolishes pleuro pericardial friction while a pericardial rub is unaltered

Hippocratic succussion is a splashing sound which can be heard when a patient who has both gas and fluid (usually pus) in the pleural cavity is shaken or moves suddenly

Post tussive suction is a sucking noise resembling that produced by an india rubber ball that has been compressed and is springing open again which is sometimes heard immediately after a cough It occurs over a cavity in the lung when its walls are not too rigid and is caused by the re-entry of the air When distinctly heard it is of considerable diagnostic value as it can only occur when a cavity is present

VI THE SPUTUM

Naked-eye Inspection The following are the principal points to be observed with the naked eye —

1 Quantity
2 Consistency
3 Whether homogeneous or in layers of different appearance
4 Colour and transparency
5 Odour

These qualities depend on the character of the material which is coughed up The main varieties are mucous sputum serous sputum purulent sputum and bloody sputum There is often a mixture of two or more types

Mucous sputum is characteristically present in early bronchitis It is clear tough and sticky As a rule the amount is not great At a later stage of bronchitis the mucus is mixed with pus cells The sputum is then less tough more copious and has a greenish yellow colour

Muco-purulent sputum occurs in many diseases of the lung In tuberculosis with cavity formation there is often found small ragged lumps of muco pus surrounded by mucus which are heavier than the other constituents since they are airless They sink to the bottom and become more or less flat and button like Thus constitutes the nummular' sputum of phthisis If there is serous or watery fluid mixed with such sputum it gradually settles into three layers the lowest being purulent the next serous and the uppermost frothy mucus Such sputum may be seen in bronchiecta is

Sputum of pus alone comes from an abscess or an empyema which has ruptured into the air passages

Serous sputum occurs apart from mucous expectoration as a thin watery fluid generally blood stained It indicates œdema of the lung Pulmonary œdema without extravasation of blood yields a white frothy sputum like soapy water

Blood may be coughed up alone or the sputum may be more or less blood stained It must be distinguished from blood brought into the mouth from epistaxis gastric hæmorrhage or bleeding from varicose veins in the walls of the œsophagus Its brighter colour and its frothy appearance usually make its origin obvious Further patients who have had a hæmoptysis commonly bring up blood stained sputum for a day or two while bleeding from the upper intestinal tract is characteristically followed by melæna Hæmoptysis may be due to pulmonary causes including tuberculosis bronchiecta is pulmonary embolus and carcinoma to cardiac causes including mitral stenosis and to aneurysm of the aorta

Several diseases cause a characteristic coloration of the sputum In pneumonia it may be rusty and so viscid that it often will not fall out of an inverted spittoon it is bright yellow or green when a liver abscess has ruptured into the lung and the latter colour also appears in some cases of pneumonia When an amœbic hepatic abscess has discharged by the lung the sputum has the appearance of anchovy sauce Black sputum is common with coal miners whilst red streaked sputum is suggestive of tuberculosis

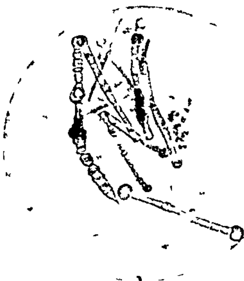


Plate 19 ASBESTOSIS BODIES FROM THE LUNG JUICE IN A CASE OF CHRONIC INDUSTRIAL ASBESTOS PNEUMONOKONIOSIS (×600)

The **quantity** of sputum coughed up in twenty four hours i important and especially whether change of position produce a large quantity or whether the sputum is produced in small amount in any position

Occasionally small **casts of bronchi** are to be found in the sputum but these are best seen with a miscroscope

The odour of the sputum is seldom very characteristic Ordinarily it has a stale smell but in cases of gangrene of the lung of fetid bronchitis and of bronchiectasis it may develop an exceedingly penetrating putrid odour An unpleasant odour may also be acquired during its transit through the mouth

MICROSCOPICAL EXAMINATION OF SPUTUM

An unstained and fresh specimen should be examined first Thereafter special methods are used for the recognition of bacteria (*see* p 406) Mixed with mucous exudation which is the basis of the sputum may be seen various organized structures as follows —

1 **Cellular structures** — i **Pus cells** in various stages of granular degeneration

ii **Epithelium** from the mouth air passages and alveoli The latter may exhibit a very characteristic iron containing pigment which is unusually abundant in cases of heart disease with pul monary congestion

iii **Red blood cells** —A few are of no importance

iv **Eosinophil cells** occur in asthma

2 **Elastic fibres** indicate destruction of lung tissue whether from phthisis gangrene or abscess

3 **Parasites** —In hydatid disease the sputum may contain hook lets more often it may contain fragments of the laminated ectocyst

4 **Asbestosis bodies** —Workers in asbestos may suffer from asbestos pneumonokoniosis In this condition the sputum contains charac teristic golden yellow bodies (Plate 19) They vary in size and shape but characteristically have bulbous enlargements at the extremities with a segmented body resembling dumb-bells The segments vary in size and the bodies vary in length from 20 to

over 200 microns An asbestos fibre forms the core and can frequently be detected They are best seen with an oil immersion lens and show up clearly without staining as golden yellow structures They can be stained by hæmatoxylin Further the golden yellow material covering each fibre contains an iron substance which gives the Prussian blue reaction when potassium ferrocyanide and hydrochloric acid are used

VII X RAY EXAMINATION

The importance of X ray examination of the chest is very great In the early diagnosis of tuberculosis and carcinoma it is paramount Serial X rays form an integral part of the estimation of progress in many chest diseases A brief outline of the standard methods will be given here

1 Radiography (Postero anterior and lateral films)
2 Screening
3 Bronchography
4 Tomography

1 The ordinary standard X ray film of the chest is a **postero-anterior** view that is to say one taken with the film against the front of the patient s chest and the X ray tube two metres behind the patient It is examined systematically on a viewing box in every case The following is a simple plan of examination

(1) **The bony skeleton** —Is the chest symmetrical ? Is any scoliosis present ? Are the ribs unduly crowded or widely spaced in any area ? Are cervical ribs present ? Are the ribs eroded or do they appear the site of malignant deposits ?

(2) **The position of the patient** —Is the patient straight or rotated ? If straight the inner ends of the clavicles will be disposed symmetrically with reference to the vertebral column

(3) **The position of the trachea** —This is seen as a dark line representing the air within the trachea The cartilagenous rings are not visible Is it centrally placed or deviated to one or other side ?

(4) **The outline of the heart and mediastinum** —Is this normal in size shape and position ?

(5) **The diaphragm** —Can the outline of the diaphragm be seen

on each side and is it normal in shape and position? Are the cardiophrenic and costo phrenic angles clearly seen?

(6) **The lung fields** —For radiological purposes these are divided into three zones —

Zone i (*Upper Zone*) extends from the apex to a line drawn through the lower borders of the anterior ends of the 2nd costal cartilages

Zone ii (*Mid Zone*) extends from this line to one drawn through the lower borders of the 4th costal cartilages and contains the hila of the lungs

Zone iii (*Lower Zone*) extends from this line to the bases of the lungs

Each zone is systematically examined on the two sides and any area which appears abnormal is carefully compared with the corresponding area on the opposite side The minor interlobar fissure which separates the right upper and middle lobes may sometimes be seen running horizontally in the 3rd and 4th interspace on the right side The major interlobar fissure which separates the lower lobes from the remainder of the lungs is not seen in a normal postero anterior film

Lateral views show whether an accumulation of fluid is situated in the anterior or posterior part of the chest and where a needle should be inserted to tap it They also show that lesions which appear at or near the hilum in the postero-anterior view are in fact situated posteriorly in the upper part of the lower lobe

The following is a simple plan of examination —

(1) **The bony skeleton**

(2) The position of the **trachea** This is seen again as a dark line running downwards and slightly backwards in front of the upper dorsal vertebræ towards the hila of the lungs

(3) **The diaphragm** —As the level differs on the two sides a double outline may be seen that of the side nearer the film being the clearer

(4) **The lung fields** —These are obscured by two relatively opaque areas one above and behind due to the shoulder joint and one below and in front due to the heart which rests on the anterior part of the diaphragm There are thus left two relatively clear areas—one above and in front behind the upper part of the sternum and one below and behind including the angle between the diaphragm and the spine

In the lateral views the interlobar fissures are more often seen Their normal positions have already been described (p 180) Their

recognition is useful both in localizing lesions and in detecting shrinkage of a lobe or lobes from fibrosis or collapse

2 Screening —This procedure is useful to detect abnormalities of movements of heart and diaphragm The patient can be easily moved in any direction

3 Bronchography —For this purpose a radiopaque iodized oil (Lipiodol or Neohydriol) is introduced into the trachea and allowed to run into the bronchi X ray pictures are taken and the corresponding bronchi are clearly outlined By means of suitable manipulations the whole of the bronchial tree can be outlined but more than one sitting may be required

The oil may be introduced

(1) by passing a special needle through the crico thyroid membrane after suitable anæsthetization of the needle track and the mucous membrane of the trachea This method is the one most usually adopted nowadays

(2) by means of a catheter passed through the nasal cavity into the trachea

(3) directly into the larynx over the back of the tongue

(4) through a bronchoscope This method is used only occasionally and for special purposes

A simple and safe method which is usually quite successful in filling the lower lobes and can sometimes be used to fill the upper one is as follows —

The patient is given 10 grains of potassium iodide in water t d s for three days and a subcutaneous injection of 2 minims of 5 per cent cocaine solution to test his sensitivity to these substances On the following day the fauces and pharynx are thoroughly anæsthetized by spraying and swabbing with 2 per cent cocaine or 2 per cent Butyn This is continued until the patient cannot swallow if he tries When this has been achieved it will be found that oil dropped blindly over the back of the tongue runs into the larynx

While the oil (10 to 20 c c preferably of Neohydriol Viscous) is being introduced the patient should sit in a chair with his body inclined towards the side which it is desired to outline Immediately the oil has been introduced he should lie on this side If one desires to outline the bronchi of the upper lobe his head

should be lowered and feet raised At the same time the patient should be rolled for a few minutes so that he lies first with his face and then with his back to the couch When it is judged that the oil has entered the bronchi films are taken in the postero anterior and lateral positions The patient must be warned not to cough from the time the oil is introduced till the examination is completed

If the bronchi on both sides are to be outlined at one sitting the side thought to be abnormal should be filled first and postero-anterior and lateral views taken The opposite side can then be filled and a further postero anterior view taken The patient should be warned not to attempt to swallow food or drink till the effect of the cocaine has worn off

4 **Tomography** —An ordinary X ray picture consists of shadows at all depths in the chest superimposed on one another It has the disadvantage that not more than some 40 per cent of the lung tissue is shown without its being obscured by shadows of the bony thorax or of mediastinal contents The tomograph is a device whereby a picture is obtained of a section of the thorax at any given depth The tube and plate are moved in the arc of a circle as the exposure is made in such a manner that the structures in one section only remain in focus and anything out of the plane of this section is blurred out Sections can be taken at different depths in the chest as desired and so the appearances in chest X rays can often be greatly simplified

VIII BRONCHOSCOPY AND THORACOSCOPY

By means of the bronchoscope the main bronchi and their branches can be directly inspected small portions of tissue can be removed for biopsy and therapeutic procedures carried out After an artificial pneumothorax has been induced the pleura can be inspected with the aid of a thoracoscope and further assistance in diagnosis may be obtained These are specialized surgical techniques

CHAPTER VII

THE URINE

Collection of samples —For most purposes a specimen of urine as passed into a clean glass vessel is sufficient If however albumin pus or blood is found in the urine of a female patient it is essential that a specimen withdrawn by catheter should be examined to exclude contamination from vaginal discharges When urine is required for bacteriological purposes a catheter specimen is essential in the case of women and in the case of men a specimen should be taken with sterile precautions after the glans penis and urethral orifice have been carefully cleaned with an antiseptic solution If orthostatic albuminuria is suspected the patient should empty the bladder immediately before getting into bed at night and pass another specimen immediately on rising in the morning

Any suspended matters soon settle to the bottom of the glass and the examination of the sample may then be conducted (1) physically (2) chemically (3) microscopically

I PHYSICAL EXAMINATION

Attention should be paid to the following points viz (1) quantity (2) colour and transparency (3) specific gravity (4) naked-eye characters of the deposit

1 **Quantity** —A healthy adult male passes on an average 50 oz. (1 450 ml) of urine in twenty four hours women a few ounces less

Normally very much more urine is secreted during the day than during the night the average volume of the night urine in a healthy adult being less than 15 oz. with a specific gravity of about 1032 The normal proportion of day urine to night urine is 100 25–60 Approximation of the night quantity to that of the day is always abnormal and is especially apt to occur in renal failure of which it may constitute one of the earliest signs Thus nocturnal frequency of urine—a symptom which is now often referred to by the

unfortunate term **nocturia** —may be the first symptom of failure of the concentrating power of the kidney and may indicate commencing renal failure

An *increased secretion* of urine occurs physiologically after increased consumption of food or drink and after exposure to cold Conversely *diminished secretion* occurs when little food or drink has been taken and after exposure to heat—especially if followed by sweating

A *pathological increase* in the urine occurs when loss of concentrating power results from any form of renal disease Polyuria is also a feature of both types of diabetes and accompanies the elimination of œdema fluid *Abnormal diminution* of urine follows sudden *lowering of arterial pressure as in shock* but is more commonly due to reduction of glomerular blood flow by inflammation as in acute nephritis or by dehydration as in fever diarrhœa vomiting and terminal heart failure With severe disturbance of renal circulation anuria may result

2 **Colour and transparency** —Normal urine is said to have [illegible] colour of amber or pale sherry The exact tint fluctuates [illegible] even in health depending upon the degree of dilution and [illegible] reaction An acid urine is always darker than one which is [illegible] even when they are equally concentrated

Small quantities of blood give the urine a smoky [illegible] and larger quantities make it brownish or red Hæm[illegible] large quantities as in blackwater fever give it a dark red [illegible] colour to one that is brownish black or almost black [illegible] of bile gives it a greenish yellow to dark brown colour [illegible] is abnormally pale when it is very dilute i e with sp[illegible] of 1002 or 1003 and in renal failure when the norm[illegible] matter (urochrome) is greatly diminished or absent

The table on p 214 shows the chief varieties of [illegible] colour of the urine with their causes

Normally when freshly passed urine is quite tr[illegible] it may be opalescent from the presence of vario[illegible] suspension If the opalescence persists after filt[illegible] to the presence of bacteria Urine that has coole[illegible] from the presence of urates In this case it will b[illegible] when warmed

Alkaptonuria —This is a very rare condition in [illegible]

ALTERATION IN COLOUR OF URINE

Colo r	C use	Cond tion or remarks
NEARLY COLOURLESS	i Large amount of urine excreted	Much drinking Nervous conditions diabetes in sipidus etc
	ii Diminution of pigment	Renal failure
ORANGE COLOURED	i Small amount of con centrated urine	Hard muscular work Fevers
	ii Increased pigment	Fevers
	iii Occasionally bile pig ment	Jaundice
ORANGE COLOURED REDDISH BROWN	Administration of rhubarb senna chrysophanic acid	(These turn yellow with acid red with alkali a normal colour going red with alkali = phenol phthalein)
ORANGE TO PINK	Phenylindanedione Dindevan	
BROWN	Bile	Jaundice
DARK BROWN	Methæmoglobin	(p 222)
RED OR PINK	i Blood	(p 221)
	ii Aniline dyes in sweets	
	iii Excessive consumption of beetroot Anthocyanuria	
PORT WINE	Porphyria	(p 237)
BROWNISH BLACK	i Melanin	Melanotic sarcoma (Darkens on standing does not reduce Fehling s solution gives greenish brown precipitate with ferric chloride blackens with HNO_3)
	ii Much hæmoglobin	(Yields chocolate deposit)
	iii Alkaptonuria	(p 213)
GREENISH BLACK	i Hydroquinone carbolic acid salol guaiacol resorcin naphthalin etc	
	ii Bile	In old standing cases of obstructive jaundice
YELLOWISH GREEN GREEN	i Bile	Jaundice (p 227)
	ii Santonin	(Turns red with alkali)
YELLOWISH AND MILKY	i Pus	(p 22?)
	ii Fat	Chyluria
BLUE	ii Administration of methylene blue	(Violet with alkali cuts off red and yellow in spectrum)

normal when passed but when exposed to the air becomes gradually darker from the surface downwards ultimately it may be dark brown or black It is due to the presence in the urine of dihydroxyphenyl acetic (homogentisic) acid

The addition of an alkali causes the urine to become dark at once Such urine reduces alkaline solution of cupric acid With Millon s reagent it gives a yellow precipitate and the addition of dilute ferric chloride drop by drop causes a passing deep blue colour The urine moreover will not ferment with yeast nor turn the plane of polarized light nor form an osazone

The condition is a rare anomaly attended by no symptoms beyond sometimes a slight dysuria and frequency at night It is due to an inborn error affecting the breaking down of the tyrosin linkage in the process of protein metabolism

3 **Specific gravity** —The specific gravity of urine is measured by a urinometer An ordinary urinometer is graduated for a temperature of 15 C and will record variations in a specific gravity from 1000 up to 1060

How to use the urinometer —The urine should be allowed to cool and should be placed in a tall jar wide enough to allow the urinometer to float freely without touching the sides All bubbles must be removed from the surface by means of a filter paper The urinometer should be wiped clean and placed floating in the centre of the jar The eye is then placed level with the surface of the urine and the division of the scale to which the latter reaches read off Care must be taken to read the level of the true surface of the urine not the edge of the rim which heaps up around the shaft of the urinometer

If only a small specimen of the urine is obtainable it may be necessary either to use specific gravity beads or else to add water to it in order to get enough fluid to float the urinometer The specific gravity found is then multiplied by the necessary figure according to the degree of dilution

Normal urine has a specific gravity varying from 1015 to 1025 If very concentrated the specific gravity may rise to 1035 even in health

The specific gravity is greatly increased by cooling If for example it is 1020 when passed it will rise to about 1025 when cooled to the temperature of the room This examination should therefore be made when the urine has cooled to room temperature

In normal urine the specific gravity is in direct proportion to the amount of urea and chlorides present An abundant urine of

high specific gravity is characteristic of diabetes mellitus In the latter condition the specific gravity may reach 1075 in most cases however it is between 1040 and 1045 In diabetes insipidus on the other hand the specific gravity may fall to nearly that of distilled water and this may also happen in hysteria The presence of albumin in the urine also affects its specific gravity 1 per cent increasing the specific gravity by three points

Serial examinations of the specific gravity constitute the simplest and one of the most valuable methods of observing renal function With normal kidneys the concentration of the urine varies to a considerable extent As renal failure develops the power of the kidneys to do this is lost and the specific gravity approximates more closely to 1010 (or 1006 if the urine is tested while still warm) With complete loss of concentrating power the specific gravity becomes fixed at 1010 the urine then being isotonic with the plasma The specimen passed on rising should always be tested for this purpose as it is normally the most concentrated that is passed in the day

4 **Naked eye characters of the deposit** —When voided normal *urine is perfectly clear and transparent After it has stood for* some time there appears in it a deposit of ' mucus This forms a woolly looking cloud which usually settles to the bottom of the glass but if the urine is of high specific gravity may be in the middle of the glass or even at the top If traces of blood are present in the urine the cloud of mucus has often a brownish tint

The normal urinary ingredients which may separate out in the form of a deposit visible to the naked eye are phosphates urates and uric acid

Phosphates —The phosphates of calcium and magnesium separate out if the urine is neutral or alkaline They form a colourless deposit soluble in acetic acid A deposit of pus is apt to be mistaken for one of phosphates but the former is not dissolved by acetic acid Deposits of pus and phosphates often occur together and the certain recognition of pus is often of the greatest importance Such recognition can *only* be made under the microscope

Urates —The urates of sodium potassium and ammonium may form a deposit if the urine is concentrated or highly acid They may appear

even in health when the urine cools Owing to their affinity for the urinary pigments the deposit is usually coloured being commonly red or like terra-cotta forming what is known as the *brick dust deposit* If the urinary pigment is scanty however the deposit may be merely yellowish or even colourless Deposits of urates can always be recognized because they disappear rapidly on heating the urine The heating ought to be done gradually because the urine may also contain albumin which if the urine is rapidly heated may be coagulated before the deposit of urates has had time to disappear and confusion may arise Acetic acid does not dissolve a deposit of urates On the other hand strong mineral acids such as nitric acid dissolve the deposit at once with the production of effervescence

Uric acid —This may form a scanty deposit visible to the naked eye The deposit occurs in the form of crystalline grains of a darkish brown colour and is therefore known as the *cayenne pepper deposit* When in doubt use the microscope

Amongst abnormal deposits the most important are the sulphonamides which may appear in the urine as a white crystalline deposit when they are present in high concentration

II CHEMICAL EXAMINATION OF THE URINE

1 REACTION

This is tested with litmus paper The urine is usually acid in reaction but it may be normally alkaline after meals This is sometimes known as the *alkaline tide* It reaches its height three hours after the taking of a meal Alkalinity of the urine may be due to ammonia formed by the bacterial decomposition of urea when the urine has stood for some time or when urea splitting organisms are present in large numbers This can be detected by its smell also by the fact that if the red litmus paper which has been turned blue is heated the red colour is restored owing to the ammonia being driven off

2 EXAMINATION OF THE URINE FOR CHLORIDES

Sodium chloride is the chief inorganic constituent of normal urine Small quantities of the potassium salt also occur The following is a simple test for measuring the approximate

concentration of sodium chloride in the urine The solutions required are —

20 per cent of potassium chromate solution
2 9 per cent silver nitrate solution

Method —Ten drops of urine are measured with a pipette or fountain pen filler into a small test tube The pipette is rinsed and one drop of the potassium chromate solution is added The pipette is then rinsed again and the silver nitrate solution is added drop by drop the test tube being shaken after each addition until the colour changes suddenly from yellow to brown The number of drops of silver nitrate used to reach the end point gives the concentration of chlorides in the urine expressed as grammes of sodium chloride per litre of urine A control test with distilled water should be performed to make sure that the chromate solution is not contaminated with chloride

The amount of sodium chloride per litre depends on the concentration of the urine The average amount excreted is about 12 grm in the 24 hours With urine of specific gravity of 1020 or more less than 3 grm of sodium chloride per litre suggests salt depletion This test is of great usefulness in the tropics when salt depletion from extreme sweating is common particularly in the diagnosis of heat exhaustion from other causes of collapse In temperate climates it can be employed in medical and surgical cases complicated by continued excessive sweating diarrhœa or vomiting to detect salt deficiency and during intravenous saline administration to indicate when a deficiency of salt has been restored but blood electrolyte estimations are to be preferred when available because the kidneys may under certain circumstances lose their ability to excrete salt so that the urinary findings may be misleading

3 ABNORMAL CHEMICAL CONSTITUENTS OF URINE

(1) *Proteins*

Before proceeding to apply tests for albumin it is essential that the urine should be *ab olutely clear* It may therefore be necessary to filter it If after filtering more than once the urine remains turbid bacteria are probably present and can be removed by long centrifugation or by shaking up the urine with powdered barium carbonate and filtering If the turbidity is due to urates it will

disappear when the urine is heated The following tests should then be proceeded with —

(1) **Boiling test** —This is the most satisfactory test for albumin but needs to be carried out with care

Fill a small test tube two thirds full of urine If the urine is alkaline add a small piece of litmus paper to the urine and add 10 per cent acetic acid drop by drop mixing thoroughly after each drop *until the litmus paper is just red* Incline at an angle boil the top inch over a flame holding the bottom of the tube and examine against a dark background A cloudiness indicates the presence of either albumin or phosphates Add 10 per cent acetic acid drop by drop and boil if the cloud disappears it consists of phosphates if it persists albumin is present Acid should be added drop by drop till no further precipitation of albumin occurs If there is more than a light cloud add a few more drops of 10 per cent acetic acid mix well and holding the test tube in a holder boil the whole volume The test tube is put aside for 4 hour or more for the albumin to settle This test is not very reliable if the urine is of extremely low specific gravity

(2) **Sulpho salicylic acid test** —This test for albumin is very reliable and does not require heat In a test tube place about 5 c c of urine Filter if cloudy add 6 drops of 20 per cent sulpho salicylic acid The formation of a cloud indicates the presence of albumin The cloud is seen best when looked for against a black background

Quantitative estimation of albumin —This can be done with sufficient accuracy for clinical purposes by means of Esbach s albuminimeter The principle of the method consists in measuring the depth of the coagulum produced in the urine by the addition of picric acid The instrument consists of a thick glass test tube with graduations on it from 0 up to 7

Method —Filter the urine if not already clear and if alkaline render slightly acid with acetic acid If the specific gravity be 1010 or more dilute the urine sufficiently to bring the density below that level (to 1008) This is important and is often overlooked Fill the tube with the urine up to the mark U Pour in the reagent (Appendix 10) up to the mark R Close the tube with a rubber stopper and gently invert it a few times to allow the fluids to mix Set aside for twenty four hours At the end of that time read off the level of the surface precipitate The figures on the scale represent grammes of dried albumin per litre of urine

Divide by 10 to get the percentage If the urine requires to be diluted the result must of course be multiplied the requisite number of times

The method yields only approximate results since the precipitates obtained in different urines vary in compactness and in the length of time they take to settle

Very small quantities of albumin cannot be estimated by Esbach s method as the instrument does not record less than 0 1 per cent If after the first trial the level of the precipitate is found to be above the mark 4 the urine must be diluted and a fresh estimation made

For ordinary purposes it is often sufficient to express the amount of albumin present as a faint trace a trace a cloud a heavy cloud or if there is enough to form a precipitate to express the height of the precipitate as a fraction of the height of the whole column of urine when the whole tube has been boiled and the precipitate allowed to settle If the fraction is one half the urine contains about 1 per cent of albumin and if one eighth about 0 2 per cent A cloud of albumin represents only about 0 02 per cent

Albuminuria may be found in disease of the kidneys or any other part of the renal tract For its significance textbooks of medicine must be consulted but a few relevant points may be mentioned here Albuminuria is of no significance in the female unless a catheter specimen has been examined If kidney disease is suspected but albumin is absent on repeated chemical examinations and blood pus or casts are not found on repeated microscopical examinations there is no renal damage and elaborate renal efficiency tests are superfluous If albumin is present in the urine in the absence of other signs of disease in the urinary tract it may be due to benign postural or orthostatic albuminuria In this condition albumin is secreted in the upright but not in the horizontal position The patient should therefore empty the bladder immediately before he gets into bed and a specimen should be passed as soon as he rises in the morning If there is no albumin in this specimen the presence of albumin in other specimens passed during the day is of no pathological significance

Bence Jones protein and allied proteins are usually but not invariably found in the urine of patients with multiple myelomatosis a fatal disease involving the hæmatogenous elements of the bone marrow This protein is also found very rarely in the urine of patients with leukæmia It coagulates at a lower temperature (55 C and under) than the usual coagulable proteins of urine Typical Bence Jones protein has also the remarkable property of going into solution again at about 100 C if

as is usually the case the concentration of salts in the urine is suitable Typical Bence Jones protein may be detected even in the presence of considerable albumin by boiling the urine which has been made slightly acid with acetic acid and filtering hot using a funnel with a hot water jacket If typical Bence Jones protein is present the filtrate will become cloudy as it cools

(2) *Blood and its Derivatives in Urine*

Blood may appear in the urine as a whole (hæmaturia) or blood pigment may appear without corpuscles (hæmoglobinuria) These two conditions can only be differentiated by examining the deposits for blood cells The detection of small numbers of red cells such as may be found for several days after an attack of renal colic can only be achieved by microscopic examination (*see* p 240)

If urine contains only a small amount of blood or blood pigment it has a peculiar opaque appearance to which the term smoky is applied Large quantities of blood give to the urine a red colour varying in intensity with the amount of blood present The blood corpuscles are apt to settle at the bottom producing a flocculent deposit which is brown or red according to the amount of the blood and the degree of its alteration

The following tests depend upon the presence of blood pigment and therefore give a positive reaction both in hæmaturia and in hæmoglobinuria —

1 **The spectroscopic test** —The following directions should be followed in all spectroscopic examinations of the urine —

1 Use a small direct vision spectroscope
2 Examine the urine in a 6-oz conical glass This permits of the inspection of layers of different thickness
3 Hold the slit 1 in from the glass and move it up and down the entire length of the cone
4 Either daylight or artificial light may be employed
5 If in doubt as to the bands shake up 400 ml of the urine with 50 or 60 ml pure amyl alcohol and a few drops of acetic acid Collect the layer which floats Clear it if need be by the addition of a little ethyl alcohol filter and examine

The urine if very dark in colour should first be diluted and it should always be filtered It should then be examined in a layer of 5 cm thick The spectrum of oxyhæmoglobin is readily detected The spectroscopic is a certain test for hæmoglobin if positive but unless that substance is

present in fair amount it may not be possible to identify the spectrum with the ordinary direct vision spectroscope (Plate 18)

ii Guaiac test —Take 1 in of urine in a test tube add to it two drops of tincture of guaiac A white precipitate forms owing to partial precipitation of guaiac resin Now add 1 in of ozonic ether without shaking If blood pigment is present a blue colour appears at the line of junction of the fluids The tincture of guaiac must be fresh and the ozonic ether should give off bubbles of gas when poured into the test tube If these points are not attended to the test may fail Sanitas is a very good substitute for ozonic ether in the above test and is less expensive

The blue colour is due to oxidation of the guaiac by oxygen derived from the ozonic ether the blood pigment acting as the carrier Ozonic ether is a solution of hydrogen peroxide in ether

Fallacies —If iodides are present in the urine a blue colour is produced on applying the test It is distinguished from that due to blood by the fact that it appears much more slowly and by its appearing simultaneously all through the fluid not at the junction of the ether and the urine

Pus in considerable quantities gives a greenish ring between the ozonic ether and the urine but microscopical examination of the urinary deposit is the only reliable method of detecting pus

Methæmoglobinuria —Methæmoglobin may be formed from hæmoglobin in any acid urine containing blood after it has stood for some time Not infrequently however methæmoglobin is present in the urine when passed It has been said to indicate that the hæmorrhage has its origin in the kidney The characteristic smoky tint of the urine in hæmaturia of renal origin is largely due to methæmoglobin and the pigment present in paroxysmal hæmoglobinuria consists mainly of it also Spectroscopic examination is the only satisfactory test for methæmoglobin It gives a band visible in the red in addition to two bands nearly in the position of those due to oxyhæmoglobin (*see* Plate 18 facing p 177)

Porphyrinuria —Porphyrins occur normally in the urine in very small amount and may be considerably increased without affecting its colour When present in large quantities the urine has a dark port wine colour Such a urine does not give the guaiac reaction If examined with the spectroscope in a thin layer it may possibly show the characteristic spectrum of so-called alkaline hæmatoporphyrin that being the form met with even in acid urines Often however no distinct spectrum can be obtained on direct examination of the urine In such a case the pigment can be extracted by shaking up the urine with a little amyl alcohol or acetic ether

after the addition of a few drops of acetic acid The extract so obtained shows the bands of alkaline hæmatoporphyrin viz four bands one at the junction of the red and yellow a second in the yellow a third in the green and a fourth (the broadest) between the green and the blue On adding a drop or two of hydrochloric acid the bands of acid hæmato porphyrin are obtained viz two bands one in the orange (narrow) and one at the junction of the yellow and green (broader) (*see* Plate 18 facing p 177) The latter is the characteristic band and consists really of two halves—a lighter half on the side next to the narrow band and a very dark half on the side away from it

Porphyrins sometimes appear in large amount in the urine of patients who are taking sulphonal or allied drugs but very much more commonly in females than in males This is a sign of very grave significance as such cases often terminate fatally The excretion of port wine coloured urine by a patient who is taking sulphonal is always an indication for the im mediate stopping of the drug and for the free administration of alkalis They are also found in rare inborn errors of metabolism known as porphyria

Urine which contains blood or hæmoglobin contains also of course some albumin and it is often difficult to say whether the blood is sufficient to account for all the albumin present or whether true albuminuria exists as well If human blood is added to normal urine in an amount sufficient to produce distinct smokiness the quantity of albumin amounts to merely a trace Even when the quantity added is sufficient to render the urine distinctly red the amount of albumin is only ½ per 1 000

(3) *Sugars in the Urine*

The sugars which are of most practical importance in urine are glucose and lactose Lævulose may sometimes occur along with glucose Under rare conditions pentoses may also occur

Glucose in the urine —Glucose (dextrose or grape sugar) $C_6H_{12}O$ is by far the commonest variety of sugar met with The condition is spoken of generally as glycosuria This must be distinguished from diabetes Diabetes—or more correctly diabetes mellitus —is a disease of which glycosuria is the chief sign but every patient with glycosuria has not necessarily got diabetes Traces of glucose occur in normal urine but not in an amount capable of detection by the reagents usually employed If therefore glucose is detected

by any of the tests we are about to describe its presence may be regarded as pathological

i Fehling s test —To prepare Fehling s solution mix equal volumes of (a) and (b) (Appendix 8) Take 1 in of urine in one test tube and 1 in of Fehling s solution in another Boil both tubes (Fehling s solution that has been kept for some time occasionally reduces itself when boiled if this should happen a fresh solution must be prepared) Add the urine to the Fehling s solution and allow the mixture to stand till cool before deciding that the reac tion is negative The test will detect about 0 2 per cent of glucose in the urine

If the urine contains 1 per cent or more of glucose a reddish or yellow precipitate appears at once on mixing the boiling urine and Fehling s solution If under 0 5 per cent is present nothing but a greenish deposit may appear on standing Slight colour changes without the formation of a precipitate should be ignored

Certain *precautions and fallacies* in the use of Fehling s test have to be mentioned

The urine must be free from albumin If necessary add a drop or two of acetic acid to the urine boil and filter

If the amount of glucose present is more than is required for reduction of all the cupric oxide some of it is apt to be caramelized especially on prolonged boiling The whole liquid and precipitate then becomes of a dark brownish colour

The fallacies attendant upon the use of Fehling s test are due to the fact that other substances in the urine besides glucose can reduce cupric oxide The chief of these among the normal ingredients is uric acid of the abnormal con tituents the chief are lactose glycuronic acid pentose and the products of certain drugs—e g chloral, chloroform aspirin and salicylates carbolic acid and ascorbic acid "Alkapton urines and *those to which formalin has been added also reduce Fehling s solution* In a doubtful case if the specific gravity of the urine is high it should be reduced by the addition of water to about 1015 Any reduction of Fehling s solution then obtained after boiling for ten seconds either immediately or on standing for a minute or two almost certainly indicates the presence of sugars in pathological amount provided the patient is taking no drugs

ii Benedict s test —To 5 ml of the reagent (Appendix 9) add 8 drops of the urine boil for two minutes and allow to cool If a reducing substance is present a precipitate will appear varying

from a light green turbidity to a red precipitate If the reduction is due to glucose the test gives approximately quantitative results —

A light green turbidity = 0 1 to 0 5 per cent of sugar
A green precipitate = 0 5 to 1 0
A yellow precipitate = 1 0 to 2 0
A red precipitate = 2 0 per cent sugar or over

Apart from homogentistic acid Benedict s solution is only reduced by glucose lactose or pentose In cases where doubt exists as to the nature of the reducing substance present two further tests can be employed—the making of an osazone with phenyl hydrazine and fermentation of the urine with yeast

A convenient simple and easily portable modification of Benedict s test which requires no external source of heat has been introduced under the name of Clinitest The reagents are contained in a tablet which is dropped into a measured amount of urine and the necessary heat is produced by the reaction of sodium hydroxide and citric acid The results agree closely with those obtained by the Benedict test

iii **Phenyl hydrazine test** —To 10 ml of the protein free urine in a test tube add 6 drops of glacial acetic acid enough solid phenyl hydrazine hydrochloride to cover a shilling and twice this amount of solid sodium acetate Heat to dissolve and filter into another test tube Immerse this in a boiling water bath for forty minutes Turn out the flame and allow the tube to cool in the bath for an hour Place a drop of the deposit on a slide cover with a cover-slip and examine under the low power of the microscope

When glucose is present bright sulphur yellow needle shaped crystals will be found arranged in tufts sheaves or rosettes If sugar is absent, only brown or yellowish globules or granules are seen, and in such a case the reduction of the Fehling s solution cannot have been due to glucose Glycuronic acid lactose and the pentoses however yield crystals which might be mistaken for those given by glucose If the nature of the reducing substance is still in doubt one should proceed to the fermentation test

iv **Fermentation test** —This test is only given by glucose and lævulose

The following precautions must be observed in carrying out the test (*a*) The urine must be acid Alkaline urine would putrefy therefore render it acid, if necessary by adding acetic acid (*b*) Boil the urine for ten minutes to drive off any air it may contain and kill bacteria cool

Use baker s yeast Shake the urine up with a small piece of it so as to form an emulsion free from lumps then place the urine so prepared in a tube Special fermentation tubes are manufactured If one of these is not obtainable an ordinary test tube inverted in a bath of mercury will do A Doremus (Southall s) ureometer tube does extremely well The long limb should be fill d with the urine completely no air bubbl s being left Set aside the tube in a warm place and examine after a few hours If a distinct bubble has appeared at the top of the tube the urine is fermentable and contains at least 0 5 per cent of glucose or lævulose The yeast must be active It should be tested with a dilute solution of glucose It is also well to have a control tube full of normal urine to which yeast has been added as the yeast is apt to give off a little gas

If these precautions are observed the test is trustworthy and delicate

The presence of lævulose in the urine may be detected by Selivanoff s reaction Dissolve 0 05 grm of resorcinol in 100 ml of concentrated hydrochloric acid and dilute with 100 ml of distilled water To 5 ml of the reagent add a few drops of the urine and heat If lævulose is present a red colour appears

Lactosuria —Lactose is sometimes found in appreciable quantity in the urine of women who are lactating and in late pregnancy It reduces Benedict s solution and gives with the phenyl hydrazine test yellow rosettes of phenyl lactosazone which are smaller than the sheaves yielded by glucose but it gives no reaction with the fermentation test

Pentosuria —This rare condition consists in the presence in the urine of pentoses i e carbohydrates containing only 5 atoms of carbon

The best test for pentoses is with Bial s reagent (1 grm of orcin 500 ml of HCl specific gravity 1151 25 drops of 10 per cent solution of ferric chloride) Boil 5 ml of this reagent in a test tube *remove from flame* add 5 drops of urine A green ring at the junction is diagnostic of pentoses Glycuronic acid and compound glycuronates give the tests for pentoses including Bial s but there is no risk of confusion if the test tube is first *removed from the flame*

Pentoses occasionally occur after the ingestion of certain fruits (cherries grapes plums) But their chief interest is in connection with so-called " pentosuria a rare anomaly of metabolism not necessarily attended by morbid symptoms probably harmless and needing no treatment

In practice if a reducing substance is present in the urine and the patient has symptoms of diabetes mellitus no further investigation is necessary If no symptoms are present it is often simplest to resort immediately to the sugar tolerance test (p 178) If the blood sugar curve is normal the reducing substance in the urine is of no pathological significance

(4) *Bile in the Urine*

Both bile pigment and bile salts may be present Usually they occur together but the pigment much more abundantly than the salts The usual cause of the entrance of the bile constituents into the urine is some obstruction in the bile passages

Urine which contains bile is greenish or brownish yellow in colour The simplest test for the presence of bile in the urine consists in shaking it in a test tube and observing the colour of the froth If this is yellow bile pigments are present in the urine Of the various other tests that have been devised the following are useful for clinical purposes —

i **Gmelin s test** —Filter some of the urine through an ordinary filter paper Then place a small drop of yellow nitric acid on the paper A spreading ring of colours from within outwards yellow red, violet and green appears around the area touched by the acid If a green colour is seen for certain bile is present in the urine

ii **Iodine test** —If a 10 per cent alcoholic solution of iodine is poured on the top of the urine in a test tube an emerald green layer appears where the two fluids join if bile is present

iii **Methylene blue test** —Place a few drops of 1 per cent methylene blue solution in a test tube and pour it away so as to leave only a trace of the dye on the surface of the tube Add 2 in of urine A green colour indicates the presence of bile pigment This is an extremely sensitive test *A normal urine should be treated similarly as a control*

Tests for bile-salts —The simplest test is **Hay s sulphur test**

Sprinkle some powdered sulphur upon the surface of the urine If bile salts are present it will sink with normal urine it floats This test depends upon the fact that bile salts lower the surface tension of fluids in which they are dissolved

(5) *Urobilinogen and Urobilin in Urine*

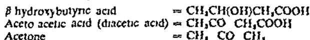
Bilirubin secreted by the liver into the bile is reduced by the bacteria of the intestine to urobilinogen and urobilin Some of this is reabsorbed and circulates in the blood stream Of this a small amount is excreted in the urine but the majority under normal circumstances is re-excreted by the liver into the bile Estimation of the urobilinogen and urobilin in the urine may therefore be

useful in several circumstances In obstructive jaundice a complete absence of urobilinogen and urobilin indicates that the obstruction is complete and that no bile pigment is reaching the intestine In patients without jaundice an excess of urobilinogen and urobilin in the urine is due to the inability of the liver to excrete these substances into the bile and indicates hepatic dysfunction Such an excess is often present in the pre icteric stage of infective hepatitis and in diffuse diseases of the liver such as severe cirrhosis

Method —To 10 ml of fresh urine add 1 ml of Ehrlich's aldehyde reagent (para dimethylaminobenzaldehyde 2 grm dissolved in hydrochloric acid 5 per cent 100 ml Pure analytical hydrochloric acid must be used) After 3 to 5 minutes normal urine shows a faint but distinct reddish tinge much intensified by heating If no red colour develops even on heating urobilinogen is absent from the urine if there is a distinct red colour in cold urine it is present in excess Normal urine shows no reddish tinge if diluted ten times with water When excess is present its degree may be estimated roughly by determining the highest dilution of urine which will produce a reddish tinge

(6) *Acetone Bodies in the Urine*

Hydroxybutyric acid aceto acetic acid and acetone may all occur in the urine in conditions of ketosis The relationship between the three may be seen from the formulæ



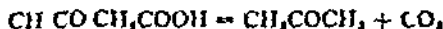
β hydroxybutyric acid	$= CH_3CH(OH)CH_2COOH$
Aceto acetic acid (diacetic acid)	$= CH_3CO\ CH_2COOH$
Acetone	$= CH_3\ CO\ CH_3$

β hydroxybutyric acid is formed first it then becomes oxidized yielding aceto-acetic acid



$$CH_3\ CH(OH)CH_2COOH + O = CH_3CO\ CH_2COOH + H_2O$$

The aceto-acetic acid is very easily decomposed into acetone and CO_2



$$CH_3\ CO\ CH_2COOH = CH_3COCH_3 + CO_2$$

It is even asserted that acetone only appears after the urine has been passed

Test for acetone bodies —The urine must be fresh and unboiled as aceto-acetic acid readily decomposes Rothera's nitro-prusside test is performed first as follows —

Ten ml of the urine are saturated with ammonium sulphate by adding an excess of the crystals 3 drops of a strong freshly prepared solution

of sodium nitro-prusside and 2 ml of strong ammonia are then added A fine permanganate colour is produced This test is given both by acetone and aceto acetic acid but by no other substances that may occur in fresh urine

If Rothera s test is negative acetone bodies are absent if positive aceto acetic acid may be tested for by the **ferric chloride reaction (Gerhardt s test)** as follows —

Take some urine in a test tube and add 10 per cent ferric chloride *solution drop by drop A precipitate usually forms and disappears again* on adding more ferric chloride The solution becomes brownish red if aceto acetic acid is present

Aspirin, antipyrin salol salicylates carbolic acid and some other drugs give a similar colour with ferri chloride Prolonged boiling (before adding the ferric chloride) destroys aceto acetic acid while the other substances whi h give a colour with ferric chloride are unaffected If therefore urine which has been subjected to prolonged boiling still gives the ferric chloride reaction it may be inferred that the reaction was not due to aceto aceti acid Boiling *after* adding ferric chloride destroys the colour whether this is due to aceto acetic acid or to other substances

A positive ferric chloride reaction is only obtained if aceto acetic acid is present in considerable amount If therefore the urine reacts to Rothera s test but not to ferric chloride it may be inferred that only small quantities of acetone bodies are present If both are positive the patient has a ketosis of considerable severity demanding urgent treatment

(7) *Drugs in the Urine*

Antipyrin —After its use the urine may be red and dichroic leading to the suspicion that blood is present On adding a little dilute perchloride of iron a purplish red colour develops which persists on boiling but disappears on adding an acid Urines containing antipyrin produce a partial reduction of Fehling s solution on boiling

Bromides —Add a little hydrochlori acid and a few drops of weak solution of bleaching powder Shake with chloroform and the latter becomes brownish red from the solution of the free bromine

Carbolic acid (*see also* section on Colour of Urine p 214) —The best test for it is to add a little bromine water The appearance of a whitish precipitate (tribromophenol) indicates the presence of phenol

Chloral chloroform etc may lead to the appearance of glycuronic acid which causes a partial reduction of Fehling s solution

Iodides—Acidify the urine with a little pure nitric acid and shake up with chloroform. The latter becomes of a rose red colour.

Iron—Add a few drops of nitric acid. Boil, cool and add a little 10 per cent ferrocyanide of potash. A precipitate of Prussian blue forms if iron is present.

Rhubarb and **santonin** have been referred to under *Alterations in Colour of Urine* (p. 214).

Aspirin, salicylates and salol appear in the urine as salicyluric acid. Such urines give a bluish violet colour on the addition of a little ferric chloride if large amounts are present; if small amounts only are present the colour may be reddish brown, indistinguishable from that given by aceto-acetic acid. These urines also partially reduce Fehling's solution.

Tannin gives a bluish black colour with ferric chloride.

Sulphonamides—1. Moisten a small piece of newspaper (paper made from refined pulp, filter paper or linen writing paper must not be used) with the urine to be tested, and place a drop of hydrochloric acid 1 : 4 dilution on the centre of the moistened area. The urine of persons taking a sulphonamide compound gives an orange yellow to orange colour. The urine of persons who have recently discontinued taking a sulphonamide gives a yellow colour for approximately six days after the last dose was taken.

2. To 5 ml. of urine diluted till colourless add 5 to 10 drops of Ehrlich's aldehyde reagent (p. 228). A bright yellow colour is given by urines containing sulphonamides.

III. ESTIMATION OF RENAL EFFICIENCY

When a considerable amount of kidney tissue has been destroyed the kidneys lose their power of concentrating the urine so that the specific gravity becomes fixed at 1010, under which circumstance the urine is isotonic with the plasma. The ability to excrete urea in sufficient amounts is also lost and the blood urea rises. Renal failure of this magnitude is thus readily demonstrated by serial observations of the specific gravity (p. 216) and by determining the blood urea (p. 170). Numerous tests have been proposed to demonstrate degrees of impairment of renal function short of that necessary to produce an increase in the blood urea, but none of them

are completely satisfactory The phenol-sulphone phthalein excretion test and the urea concentration test will be described here Another one often employed is the urea clearance test of Van Slyke but for this larger works must be consulted

(I) THE PHENOL SULPHONE PHTHALEIN TEST FOR RENAL EFFICIENCY

The principle of the test is to estimate the amount of the dye phenol sulphone phthalein excreted in two hours after the injection of 6 mg

In order to secure a good flow of urine 400 ml of water are given to drink *After about 15 minutes the patient s bladder is emptied if necessary* by catheter and 6 mg of the dye dissolved in 1 ml. of water are injected intramuscularly or intravenously

After exactly one hour and again after two hours the bladder is emptied, and the whole of these two specimens reserved for estimation of the dye colorimetrically in each.

The urine being usually acid, will not be coloured red by the dye until alkali is added Each specimen is measured, made strongly alkaline with 40 per cent NaOH made up to 500 ml if the red colour developed is deep to 250 ml if not deep and filtered to remove the precipitate of phosphates

To serve as a standard with which to compare the urines 1 5 ml of a 0 02 per cent solution of phenol-sulphone phthalein is made alkaline and brought up to 50 ml thus gives a concentration equal to 6 mg in 1 000 ml This standard has to be matched against a solution in urine and, in order to allow for the yellow colour of the urine urine should be used in making up the standard. The most convenient way is to make up two solutions A and B

A Add 1 5 ml. of 0 02 per cent solution to 40 ml of distilled water add 1 ml of 40 per cent NaOH and make up to 50 ml with distilled water

B Add 1 5 ml of 0 02 per cent solution to 40 ml of normal urine less if highly coloured, 1 ml. of 40 per cent. NaOH make up to 50 ml. with distilled water and filter

These solutions contain the same amount of the red dye and by mixing them in various proportions it is possible to obtain a standard containing the right amount of yellow pigment to match the urines to be investigated

Such a mixture of A and B having been made suitable for the first hour urine the standard and urine are put in the two pots of a colorimeter the standard set at 70 mm and the depth of the unknown required to give an equal depth of colour read.

Supposing this urine has been diluted to 250 ml and the reading observed is Y

Then

$$\text{Depth of colour in unknown} \times \frac{Y}{20} = \text{depth of colour in standard}$$

$$\text{amount of dye in 250 ml unknown} \times \frac{Y}{20} = \text{amount of dye in 250 ml}$$

of standard

$$= \frac{250}{1000} \times 6 \text{ mg} \quad \text{or 25 per cent of the original dose}$$

$$\text{amount of dye in the unknown} = \frac{20}{Y} \times 25 \text{ per cent of original dose}$$

if the urine was diluted to 500 ml the amount of dye it contained would be $\frac{20}{Y} \times 50$ per cent of the original dose

The percentage of the original dose in the second hour specimen is estimated in the same way

If a colorimeter is not available the estimation may be performed by dilution as follows —

In two similar test tubes of equal bore are placed 10 ml of the unknown and of the standard and distilled water is added from a burette till the solutions match

Suppose Y ml are added to the standard

Then

$$\text{depth of colour in unknown} \times \frac{10 + Y}{10} = \text{depth of colour in standard}$$

$$\text{dye in 250 ml unknown} \times \frac{10 + Y}{10} = \text{dye in 250 ml standard}$$

$$= \text{25 per cent of original dose}$$

$$\text{dye in 250 ml unknown} = \frac{10}{10 + Y} \times \text{25 per cent of original dose}$$

The amount of the original dose excreted in the first hour by a normal adult is usually over 40 per cent and in two hours together over 60 per cent

(ii) UREA-CONCENTRATION TEST

The concentration of urea in the urine taken under ordinary conditions is of little value as an index of the efficiency of the kidneys since a normal person may excrete a urine of low urea-concentration when the amount of urea formed is small or the volume

of water passed is large The patient is therefore given a test dose of urea which should if renal efficiency is unimpaired provoke a high concentration in the urine

Method —15 grm of urea in 100 ml of water flavoured with a little tincture of orange is given by the mouth when the patient has had nothing to drink for some hours and after emptying the bladder the urine passed in each of the subsequent three hours is collected and the urea-concentration estimated preferably in the specimens from the second and third hours

If the kidneys are healthy the concentration in the second and third hours is usually over 2 5 per cent and almost invariably over 2 0 per cent unless the volume is large (over 150 ml per hour) With moderate damage to the kidney concentrations from 1 5 to 2 5 per cent may be encountered and with severe damage under 1 5 per cent A certain amount of judgment is required in interpreting the result such high concentrations cannot be expected when the initial blood urea is low (e g about 0 020 per cent) as when it is comparatively high (e g about 0 045 per cent) nor such high concentrations when the volume passed is comparatively large (e g 130 ml) as when it is small (e g 50 ml)

This test is a useful complement to the phenol sulphone phthalein test as the latter sometimes gives low figures when the kidneys are little affected If low figures for phenol sulphone phthalein are accompanied by low figures for urea-concentration there is little doubt that the kidneys are deficient On the other hand the phenol sulphone phthalein test rarely gives normal figures when the renal efficiency is reduced and high figures for phenol sulphone phthalein confirm the urea-concentration test in its doubtful zone between 2 0 and 2 5 per cent

IV MICROSCOPICAL EXAMINATION OF URINARY DEPOSITS

The urine is centrifuged and a drop of the deposit is placed on the centre of a slide and covered with a cover glass The preparation is then examined with both the low (⅔ in or ⅓ in) and high (⅙ in) objectives the microscope being vertical and the diaphragm partly closed

UNORGANIZED DEPOSITS

The first group of urinary deposits includes the various salts and crystalline substances that are found in urine either when freshly

voided or more often when it has stood for some time The following occur in acid urine (Fig 53) —

1 Uric acid —This appears under a variety of forms and unless the urine is almost devoid of colouring matter assumes a reddish brown colour in consequence of its absorbing a considerable amount of pigment. To the naked eye the appearance resembles that of a shower of grains of cayenne pepper collected at the bottom of the specimen Under the microscope the crystals are either rhombic prisms or some modification of that form Often the more obtuse angles are rounded off and the edges continued in curved lines so that pointed oval shapes result Numerous crystals may be joined together to produce rosettes and other composite forms

2 Urates —These are urates of potassium sodium and ammonia They have a considerable affinity for the urinary pigments and hence are generally more or less pink or brick coloured In very pale urines they are colourless and resemble closely a deposit of phosphates Microscopically they consist of small granular particles arranged in moss like clumps Ammonium urate may form spheres either solitary or in clusters having a more or less crystalline structure with numerous spines radiating from their surface On heating a urine from which they have separated out they will be found to redissolve before the boiling point has been reached

3 Hippuric acid appears in human urine chiefly after the administration of benzoic acid or its salts It occurs as colourless four sided prisms insoluble in hydrochloric acid but soluble in ammonia

4 Calcium oxalate —This deposit is rarely abundant The small colourless crystals lying on the top of the numerous deposits at the bottom of the urine glass give the impression of an undulating snowy surface Two forms are found under the microscope. The first which is by far the commoner consists of small octahedral or envelope like crystals. The other is that of minute dumb-bells or oval biscuit shaped discs.

5 Cystin is a rare deposit in human urine but when it occurs the precipitate is often copious and is not unlike a sediment of fawn-coloured urates It is soluble in alkalis and in strong acids (such as hydrochloric) but insoluble in acetic acid It does not separate out from alkaline urine but precipitation may occur upon addition of a few drops of acetic acid It is deposited as hexagonal tablets soluble in ammonia and when the

ammonia evaporates recrystallizing as hexagons or prisms. Cystinuria is a rare congenital abnormality of theoretical importance only were it not that the cystin may form calculi in the kidneys and bladder

6 Tyrosin is generally found associated with leucin, but also occurs independently It forms colourless sheaves of fine needle like crystals A similar appearance may be presented by several other deposits if there is any doubt as to the nature of the sediment a chemical analysis may be necessary

7 Leucin occurs in urine as yellow spherical masses without obvious crystalline structure Leucin and tyrosin may be found together in acute yellow atrophy of the liver

In alkaline urine the following occur (Fig 54) —

1 Phosphates —These may be salts of phosphoric acid and calcium or of phosphoric acid with ammonium and magnesium

(a) Calcium phosphate is found either in an amorphous or in a crystalline form, the latter being also known as *stellar phosphates*

Amorphous calcium phosphate occurs in small white granules as a deposit in alkaline urine To the naked eye the sediment is white and flocculent unlike urates it has no affinity for urinary pigment The deposit is increased on heating

Stellar phosphates are uncommon They consist of colourless prismatic crystals which occur either singly or more often in radiating clusters They are found in very faintly acid as well as in neutral and alkaline urine

(b) Ammonium magnesium or "triple" phosphate —To the naked eye the sediment appears very white and when the crystals are large they may be visible as bright points Sometimes the deposit also clings to the sides of the glass and forms a film on the surface of the urine

The crystals are incomplete triangular colourless prisms which may offer considerable variations in appearance according to their length and degree of perfection Often they are described as knife rest or "coffin lid" crystals

2 Ammonium urate is very commonly present in cases of cystitis. Microscopically it occurs in small spherical masses which may have smooth surfaces, or be beset with innumerable spiny processes.

3 Carbonates generally occur as granular particles which dissolve in acetic acid with evolution of CO_2 As phosphates give off no gas on solution in acetic acid it is quite easy to distinguish between them. On



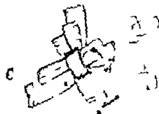
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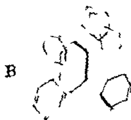
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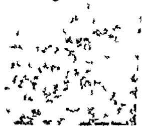
A



C



B



D

Fig 53

1 Sulphathiazole crystals in urine (× 300 approx)

2 Sulphapyridine crystals in urine (× 300 approx.)

(D wings b sed on riginal photographs ppl d by th M y and B ker Biological L bo t es)

3 Some crystals which may be seen in acid urine (× 450 approx)

A C lcium l te.
B Cy tine
C Uri acid.
D Am rph us t s.

(D wn by Dr C P F Bo ld n.)

1 2

A C D

C

B C

3

Fig 54

1 Blood fatty hyaline and granular casts in urine with red blood cells (× 300 approx)

2 Epithelial cells and pus cells in urine (× 300 approx)

3 Some crystals which may be seen in alkaline urine (× 450 approx)

A D mb bell ry tals of cal um bon t
B Ammonium b te.
C. Prism t tripl ph ph t s.
C Tripl ph sph s (feathery form in rap d prec p t tion)
D Cal um ph ph t (f intly alkalin rine)

(Drawn by D C P F B ld n.)

rare occasions in human urine and commonly in horses urine calcium carbonate appears in the form of dumb-bells or of spheres with a radiating crystalline structure

4 **Cholesterol** has occasionally been found in the urine it occurs as characteristic thin rhomboidal, colourless plates with a notch at one of the corners

Sulphonamide crystals may be found in the urine of patients taking these drugs Their form is extremely variable (Fig 53) and differs with different sulphonamides so that no short description is possible In cases of doubt the chemical reactions described on p 230 should be applied to the urine containing the suspected crystals

ORGANIZED DEPOSITS

1 **Red blood corpuscles** (Fig 54) are best sought in recently voided urine Usually of normal size they may according to the density of the urine appear either swollen or shrunken and crenated They are recognized by their shape and biconcavity and particularly by their colour Droplets of oil from a catheter are frequently mistaken for red blood-corpuscles The droplets are readily differentiated by their higher refractive index more circular shape and variable size The presence of small numbers of red cells in the urine—so-called microscopic hæmaturia, may cause no change in its microscopic appearance and give no positive guaiac reaction Its detection which can only be achieved with the aid of the microscope may be of great importance in diagnosis particularly in cases of subacute bacterial endocarditis An occasional red cell may be found in apparently normal urine More than one per ten high power fields should be regarded as abnormal

2 **Leucocytes or pus corpuscles** (Fig 54) can generally be recognized by their round shape the lobed form of their nuclei and their refractile granular appearance When numerous they may appear in clumps or groups Pus may form a deposit visible to the naked eye in the specimen glass and may give a green colour with the guaiac reaction but a certain diagnosis can only be made by seeing pus cells under the microscope An occasional leucocyte may be seen in the deposit from the urine of apparently normal persons but the presence of pus cells in any number is always pathological

One must make sure that the urine has not been contaminated and in women a catheter specimen is essential if the urine is to be examined for either blood or pus

3 **Epithelium** (Fig 54) from various parts of the urinary tract may be found in the urine

4 **Spermatozoa** occur at times in the urine where their characteristic appearance makes it easy to recognize them

5 **Prostatic threads** are found when there is chronic inflammation of the prostate especially after gonorrhœa They are much larger than tube casts being visible readily enough to the naked eye as they float in the urine or on its surface

6 **Tube casts** —The recognition of casts (Fig 54) is important as their presence in any number indicates that the patient is suffering from some form of nephritis They are first recognizable under the $\frac{2}{3}$ objective but should be further examined under the $\frac{1}{6}$ Casts are distinguished from other objects which may be mistaken for them—such as hairs wool cotton masses of urates rolled up epithelial cells and so-called cylindroids by their shape and sharply defined outline They are always cylindrical in shape and may have rounded ends or one end may be ragged as if fractured An occasional hyaline cast may be found in apparently normal urine granular or epithelial casts or hyaline casts in numbers indicate a nephritis of some variety

The following varieties are described

i Cellular —The cells may be epithelial or composed of red blood corpuscles or leucocytes

(a) *Epithelial* —The casts may be completely covered with epithelial cells as though the whole epithelium had scaled off a tubule or the cells may have been separately detached and subsequently moulded

(b) The *red blood corpuscle* casts exhibit a surface thickly covered with the minute round corpuscles

(c) *Leucocytes* rarely form casts by themselves but are fairly often found adhering to the surface of other casts

ii Granular —The granules are sometimes coarse at other times fine They are sometimes fatty at other times they result from granular degeneration of protoplasm

iii Amorphous —This group contains two varieties the hyaline and the colloid

(a) *Hyaline* tube-casts are pale transparent and homogenous Occasionally the surface is striated They may be almost invisible but are rendered more prominent by the addition of iodine solution

(b) *Colloid* casts are broader and more highly refractile than hyaline Often they are more or less fissured

iv Lipoid —These are found when the tubular cells are undergoing lipoid degeneration

Cylindroids resemble extremely long and narrow tube-casts but are usually considerably flattened, tapering and may be frayed out at the ends

Not to be confounded with either tube-casts or cylindroids are the little strings of mucus which are occasionally present in a urinary sediment Small clumps of micrococci and short so-called prostatic threads are also liable to be misinterpreted

7 Tumours of the bladder especially when villous may very rarely be detected by the presence of fragments of the growth in the urine

8 *Schistosoma hæmatobium* is the only parasite of any importance found in the urine The ova (Plate 18 19) measure 0 12 mm by 0 04 A spine projects at one pole The ova of *Schistosoma mansoni* which are found in the fæces have a lateral spine

9 Foreign bodies often occur in urine which has been set aside for examination Besides hairs feathers moth wing scales cotton woollen and silk fibres and starch grains derived from dusting powders (readily recognized by their turning blue on the addition of a little dilute tincture of iodine) one occasionally finds fragments of the contents of dermoid tumours or abscesses that have opened into the bladder or ureter

It may happen that the patient has vomited and sputum or vomited matter may be more or less abundantly mixed with the urine

CHAPTER VIII

THE SKIN

FOR the examination of the skin and its appendages the pati should be stripped as completely as circumstances permit a should be examined by daylight, ✓

First notice the colour of the skin The normal colour is v variable *some persons having a fresh complexion and othe* though quite healthy a pale one Pallor is also often seen in nourished persons in persons who work underground or indoo in those who have lived in the tropics and in a variety of illness It may be seen temporarily in hæmorrhage shock and in inter emotion Anæmic persons are often pale *but not all pale perso are anæmic* The colour of the mucous membrane of the eyeli and mouth is a better indication of anæmia than is the colour the skin *Undue redness* is seen in overheating extreme exertio sunburn some fevers and in many of the exanthemata *Cyano.* is a bluish or purplish tint which may be more or less generaliz or limited to one or more extremities It is due to the presence an excess of reduced hæmoglobin resulting from impaired ox genation or circulation of the blood and is seen in certain diseas of the heart or respiratory system in peripheral circulatory failur in polycythæmia and pituitary basophilism *It is important to no* that *methæmoglobinæmia* may produce a blue tint which is le bright and more leaden than cyanosis Methæmoglobinæmia ma be due to poisoning by aniline nitrobenzene or to drugs such a phenacetin or sulphanilamide

Jaundice varies from the sub-icteric lemon yellow c daffodil tints seen in pernicious anæmia and acholuric jaundic to various shades of *yellow orange or dark olive green in obstru* tive jaundice Jaundice must be distinguished from yellowness du to the taking of mepacrine either as a prophylactic against or trea ment for malaria in those who have recently returned from th tropics or for the treatment of lupus erythematosus and from rar cases of carotinæmia due to the presence of an excess of lipoic

soluble yellow pigments in the plasma Neither mepacrine nor carotene however stain the conjunctivæ which jaundice does Slight degrees of jaundice cannot be seen in artificial light

Normal skin contains varying amounts of brown pigment A congenital absence of pigment in the skin which may be generalized or occur in patches is known as *albinism* Alternating patches of white and darkly pigmented skin are seen in *leucomelanodermia* Increased pigmentation may be racial due to sunburn or may occur in various diseases In *Addison s disease* there is a brown or dark brown pigmentation affecting exposed parts parts pressed on by stays or corsets parts normally pigmented such as the axillæ and very characteristically the mucous membranes That of the lips and mouth should always be examined and may exhibit dark bluish black areas that have been compared with the stains produced by sucking a pen More or less generalized pigmentation may also be seen in *hæmochromatosis* where it has a peculiar bronze colour with a metallic sheen in *chronic arsenic poisoning* where it is finely dappled and affects covered more than exposed parts in *argyria* where the deposition of silver in the skin produces a diffuse slaty grey hue and occasionally in the cachexia of advanced malignant disease More localized pigmentation is seen in *pregnancy* where it particularly affects the nipples and their areolæ and the linea alba in *hyperthyroidism pellagra rheumatoid arthritis* and a variety of prolonged wasting diseases Localized pigmentation is seen in scars of various kinds particularly those due to X irradiation therapy and those following varicose ulcers of the legs Phtheiriasis or *vagabond s disease* a pigmentation due to chronic infestation with lice is now rarely seen *Erythema ab igne* a coarsely mottled pigmentation of the legs of women who habitually sit too near a fire is however common (Plates 3 and 4)

Hæmorrhages into the skin occur in various forms and in various conditions If less than 1 mm in diameter they should be referred to as *petechiæ* if from 2 to 5 mm in diameter as *purpuric spots* and if larger as *ecchymoses* If the hæmorrhage is large enough to produce an elevation of the skin it is referred to as a *hæmatoma*

Petechiæ and purpuric spots do not disappear when they are pressed on by a glass slide or lens which serves at once to distinguish them from erythematous spots such as the rose spots of typhoid fever and from *telangiectases* which consist of a small collection of dilated skin vessels They must also not be confused

with capillary nævi (de Morgan s spots) which are common and have no pathological significance

Next one should seek the presence of any **eruption** If present inquiry should be made on the lines laid down on p 8 The exact situation and extent of the eruption should be noted and whether it is symmetrical or confined to one side only One should then pass to a description of the minute characters of the eruption In order to do this it must be remembered that every cutaneous eruption consists of a **primary lesion** to which secondary lesions may or may not be superadded The following is a description of the different primary lesions which may be met with —

1 **Macules** (spots) —Any abnormal change in the colour of the skin confined to a limited area Always note whether or not they fade on pressure The rose spots of typhoid fever for example fade on pressure whilst those due to hæmorrhages into the skin do not

2 **Papules** —Solid projections above the surface which are not larger than a pea The term **tubercle** or **nodule** is applied to any solid projection from the skin which is larger than a pea but not larger than a cherry Anything larger than that is called a **tumour** Always note whether the top of a papule is rounded as in some forms of eczema pointed as in acne or flattened as in lichen As regards the base observe whether it infiltrates the skin widely or not The wider the infiltration the more extensive and severe the inflammation

3 **Vesicles** —Elevations of the horny layer of the epidermis by transparent or milky fluid which are not larger than a pea If larger than this they should be described as **bullæ** or **blebs** Always note whether or not there is an area of redness around the base of a vesicle for such redness indicates that the vesicle is planted upon an inflamed base—a fact which may be of diagnostic value

4 **Pustules** —Small elevations of the skin containing pus Always observe whether there is much infiltration around them or not

5 **Wheals** —Slightly elevated portions of skin the centre of which is paler than the periphery

Having stated which of these primary lesions it is that composes

the eruption one should next note whether the lesions are isolated (discrete) or whether they run together It must also be remembered that an eruption may be made up of more than one kind of primary lesion Thus papules may be mingled with pustules or pustules with vesicles and so on

Next look for secondary lesions These are either produced mechanically or are the result of changes which take place in the primary lesion in the course of its growth or decline The commonest secondary lesions of mechanical production are excoriations due to scratching and fissures (rhagades)—deep cracks going down to or through the corium and produced by the stretching of the skin after it has become inelastic owing to infiltration Fissures are often very painful

The following are the secondary lesions produced by changes in those which are primary —

i Desquamation —If the primary lesion is a dry one (macules or papules) a scaling off of epidermic cells occurs and the eruption is then said to be *scaly*

In moist lesions (vesicles pustules bullæ) the epidermic cells become glued together by the dried fluid and a scab or crust forms The scab may be serous purulent hæmorrhagic or sebaceous according to the nature of the contents of the primary lesion

ii Infiltration may occur around the primary lesions leading to the production of a leathery feeling in the skin This is usually the result of prolonged chronic inflammation

iii Pigmentation may occur around the primary lesions This also is usually due to prolonged inflammation

iv Ulceration —Due to breaking down of the primary lesions and destruction of a part of the true skin

The points to note in describing an ulcer are (1) the nature of the floor of the ulcer and the granulations covering it (2) the character of the edge—smooth raised undermined etc (3) the discharge whether serous purulent watery fetid etc (4) the character of the surrounding skin whether indurated pigmented etc It is also important to examine the lymph nodes that drain the area of the ulcer

v Scar formation —This only occurs where the true skin has been involved i e where there has been an ulcer Describe the scar noting especially whether it is thin or thick freely movable or adherent to the deeper tissues pale or livid pitted or not surrounded by a zone of pigmentation or not

Proceed now to the **palpation** of the skin Pass the hand gently over it pinching it up between the forefinger and thumb and note the following points —

Is it smooth or rough thin or thick dry or moist ? If there is any visible sweating note whether it is general or local whether it is attended or not by any flushing of the skin and whether the sweat has any particular odour

The **elasticity** of the skin should be investigated If a fold of healthy skin is pinched up it immediately flattens itself out again when released Sometimes however it only does so very slowly remaining for a considerable time in a creased condition This may be of little or no significance in old persons with loose inelastic skins but may be an important sign of dehydration in conditions associated with prolonged vomiting and diarrhœa

The condition of the **subcutaneous tissue** should also be investigated The presence of **œdema** is usually recognized by the fact that if the skin is pressed with the finger especially over a hard body such as a bone a pit is left which persists for some little time In some cases no pitting can be produced especially when the œdema is of very long standing The best place to look for slight degrees of œdema in cardiac disease is behind the malleoli of the tibia and fibula in patients who are ambulent and over the sacrum in those who are confined to bed The pressure of the finger should be maintained for 20 to 30 seconds or small degrees of œdema will be overlooked

Subcutaneous emphysema gives rise on palpation to a characteristic crackling sensation It starts in and is usually confined to the neighbourhood of the air passages or air-containing organs In rare cases it may be due to infection with gas gangrene organisms

Microscopical examination of the skin and its appendages is confined to the diagnosis of some **parasitic diseases** of which the following are the chief —

1 **Scabies or itch** —This is due to the *Acarus* (*Sarcoptes*) *scabiei* The female acarus is larger than the male and forms burrows in

the skin in which the eggs are deposited These burrows should be looked for between the fingers and on the inner aspects of the wrists They are recognized with the naked eye as little short dark lines terminating in a sort of shining spot of skin The eggs lie in the dark line the insect in the shining spot It may be picked out by means of a flat surgical needle passed along the black line to the clear spot The use of a lens aids the operation—which is *by no means invariably successful—and permits of the recognition* of the insect The latter may be placed on a slide under the microscope for more minute inspection

2 Pediculosis —Three varieties of pediculus occur—*Pediculus capitis* on the head *P corporis* on the trunk *P pubis* on the pubic and axillary hairs The eggs or nits of *P capitis* are stuck on the hairs From their position on the hairs one can judge roughly of the duration of the condition for they are fixed on at first near the root of the hair and are then carried up with the latter in its growth The higher up the nits are therefore the longer have the pediculi been present *P corporis* should be looked for in the seams of the clothes especially where the latter come into close contact with the skin—e g over the shoulders The bites of the parasite produce hæmorrhagic spots each with a dark centre and a paler areola Marks of scratching should always be looked for on parts accessible to the patient s nails

P corporis is the longest of the three *P pubis* is shortest and *P capitis* is between the two in size *P pubis* is also distinguished from the others by being yellowish brown in colour *P capitis* and *P corporis* are both greyish in colour though the latter varies considerably with the colour of the skin of its host The shape of the thorax and abdomen forms a distinguishing character between these varieties and *P pubis*

3 Fungus Infections —Fungus may grow in the skin nails or hair and cause disease (ringworm) *Skin*—Between the toes on the soles of the feet and in the groins are the commonest sites The lesions may be scaly and vesicular areas tending to spread in a ring form and healing in the centre scaly erythematous plaques with festooned margins areas of hyperkeratosis on the parts of the skin which have a thick horny layer (palms and soles) or macerated dead white offensive smelling epithelium in the

intertriginous areas such as the toe clefts *Nails*—Discoloration deformity hypertrophy and abnormal brittleness may result from fungus infection *Hair*—Ringworm of the scalp is most common in children It presents as round or oval areas of baldness covered with short broken off lustreless hair stumps These hair stumps usually give a bright green fluorescence when exposed to long wave ultra violet light (UVL filtered through Wood s glass)

Microscopical examination—Scales from the active edge of a lesion are scraped off lightly with a scalpel or the roofs of vesicles are snipped off with a scissors The material is placed in a drop of 10–20 per cent aqueous potassium hydroxide solution on a microscope slide covered with a cover slip and left for 30 minutes to clear It is then examined with $\frac{1}{3}$ in or $\frac{1}{6}$-in objective using low illumination The mycelium is recognized as branching refractile threads which boldly transgress the outlines of the squamous cells (mycelium which respects the cells outlines is mosaic fungus an appearance probably produced by inter cellular lipoid) Nails are examined in much the same way but as nail is harder and denser it is necessary to break up the snippings and shavings into small fragments These are either heated in potassium hydroxide or are left to clear in it overnight before being examined A scalp lesion is cleaned with 70 per cent alcohol or with 1 per cent cetrimide and infected stumps are extracted by traction in the long axis of the hair with epilation forceps (this is most conveniently done under Wood s light) The hairs are cleaned *in potassium hydroxide in the same way as skin scales* Examination under the microscope reveals spores on the outside of the hair roots and mycelium inside the hair substance The species of fungus responsible may be established by culture on Sabouraud s glucose agar or on wort agar medium

CHAPTER IX

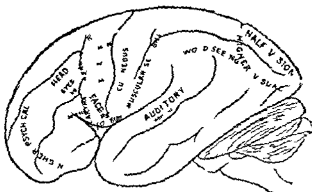
THE NERVOUS SYSTEM

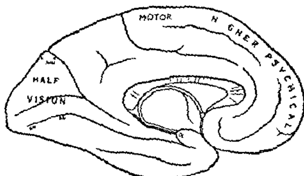
General considerations —The aim of a neurological examination is to determine the site and nature of the lesion responsible for the patient s symptoms in a case of nervous disease It is also an essential part of any routine examination for signs may be found in the nervous system in the absence of symptoms which lead to the diagnosis of conditions affecting or apparently affect ing other parts of the body Precise history taking is also impor tant as the evolution of any particular condition must be known if an accurate diagnosis is to be achieved Due regard should be paid to the previous and family histories which may yield valuable clues to the nature of the patient s illness and the examination of other systems must not be neglected for instance inspection of the skin skeletal system chest abdomen or cardiovascular system may provide information leading to the diagnosis of a nervous disorder

A detailed neurological examination is an ordeal for ill patients and a test of concentration and co-operation in those in good general health Care should be taken not to fatigue the patient unduly Overlong examination may defeat its own ends especially when sensation is being investigated by leading to variable and incongruous findings It may be necessary to conduct the examina tion in more than one session

Whilst the mode of examination outlined in the pages which follow is that generally adopted it need not be rigidly adhered to For example if a patient is complaining of sciatic pain there is no reason why one should not begin with the examination of the lower limbs and lumbar spine

Observation of the patient s ordinary activity for example the way he walks into the room and undresses for examination is often helpful The minimum record of a negative neurological examina tion should include a statement that the optic discs pupils other cranial nerves and motor and sensory systems are normal a record should be made of the state of the tendon reflexes and of the abdominal and plantar responses





2

Plate 20 (1) OUTER (2) MESIAL ASPECTS OF LEFT HEMISPHERE SHOWING FUNCTIONAL AREAS

CHAPTER IX

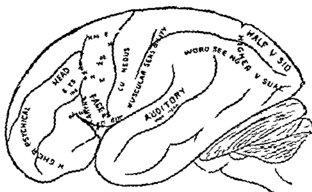
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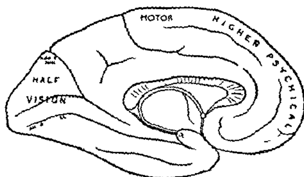
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2

Plate 20 (1) OUTER (2) MESIAL ASPECTS OF LEFT HEMISPHERE SHOWING FUNCTIONAL AREAS

(After [illegible])

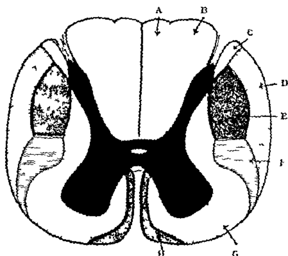


Plate 21 TRACTS OF THE SPINAL CORD

A—Goll [illegible] } Dorsal col[illegible]
B [illegible] col[illegible]
C D[illegible] lat[illegible]
D C[illegible]rebell[illegible]
E Lat[illegible] p[illegible]
F [illegible]
[illegible]
H [illegible]

ANATOMY AND PHYSIOLOGY

The **dominant hemisphere** i e that hemisphere which plays the major part in control of a person s activities especially that of speech is situated on the left side in right handed people and on the right in left handed people

The motor system (1) *The lower motor neurone*—Muscular *movement depends ultimately on the integrity of the lower motor* neurones which connect striped muscles with the central nervous system The lower motor neurones consist of the anterior horn cells and homologous cells in the brain stem and their fibres which pass via the anterior spinal nerve roots and peripheral nerves to the muscles The nerve impulses which influence the muscles pass via this final common pathway to the motor end plates and are then transmitted humorally to the muscle fibres If this path way is interrupted at any point muscular wasting and weakness hypotonia and loss of tendon reflexes occur These are the car dinal signs of a lower motor neurone lesion

Although various reflex movements operate at a spinal level the initiation of voluntary and more complex movements and the maintenance of posture and muscle tone depend on impulses arising from higher centres These impulses can only reach the muscles if the final common path is intact These higher centres consist of the pyramidal and extra pyramidal systems and the cerebellum

(2) *The pyramidal system*—This system consists of the pathways which link the cerebral cortex directly with the lower motor neurones in the brain stem and spinal cord The fibres concerned are gathered in the pyramidal tracts They arise from the motor area of the brain but the pyramidal tracts also contain fibres which arise from the post central cortex and subcortical structures The motor area of the brain occupies the anterior wall of the fissure of Rolando and the adjacent parts of the pre-central gyrus There is localization of function in the motor cortex different parts of the opposite side of the body being separately represented Those parts of the body which carry out the most skilled movements for example the fingers and thumb have the largest areas of repre sentation The areas for the tongue jaw and facial movements lie lowest in the motor cortex those for the arm trunk and leg

following successively as the motor area ascends on to the medial aspect of the hemisphere (Fig 55)

The fibres of the pyramidal tracts pass downward from their cells of origin into the internal capsule occupying the anterior two thirds of the posterior limb (Fig 56) Here the order of representation of the body is face shoulder elbow hand trunk and lower limb from before backwards The pyramidal fibres now descend to occupy the middle three fifths of the peduncles of the mid brain in the same order below this level there is no segregation of the

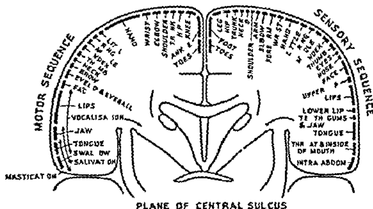


Fig 55 —Rasmussen and Penfield s diagram of localization in motor (left) and sensory (right) cortex

fibres according to their destination consequently a lesion affecting the tract does not produce paralysis of a single limb Passing through the pons the tract becomes broken into scattered bundles by the transverse pontine fibres and nuclei pontis In the upper part of the medulla the fibres join to form the pyramids which are well marked protuberances on the anterior aspect of the brain stem In the lower part of the medulla the greater number of fibres decussate with those of the opposite side and pass backwards to run down the spinal cord in the lateral columns as the crossed pyramidal tracts A smaller number of fibres do not decussate continuing downwards in the anterior columns as the direct pyramidal tracts but these eventually decussate at lower levels in the anterior

commissure *Some uncrossed fibres descend in the crossed* pyramidal tract of the same side ending in the anterior horns of the same side All the pyramidal fibres terminate at different levels in the grey matter of the brain stem or spinal cord usually

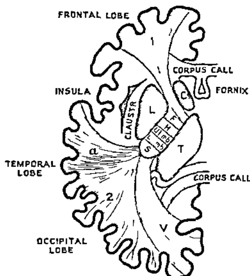


Fig 56 —Internal capsule and corona radiata

T Th lamu L Lenticul ucl us C Ca d l F Sup lear tract f be facial rve H S p ucl tra t f th hypogl sal rv U l mb Supranuclear tra t f arm m scl L lumb S pra l t t f l g m scl S Sensory acts (thal mo—cort l racts) *a* A d t ry tracts t mporal l be V sual t ct t occi- p tal l be 1 F tu p tin tract d co d ta 2 Occip to-tempo o-po tin tra t and orona radi ta.

(*After Bing*)

ending in relation with internuncial cells rather than with the *anterior horn cells themselves (Fig 57)*

The pyramidal system is concerned with the initiation of the more voluntary and skilled motor acts The paralysis resulting from lesions of the system is usually widespread as of one side of the body (hemiplegia) or of a whole limb (monoplegia) In hemi plegia due to pyramidal disease the movements of the head and trunk which are *bilaterally innervated often escape altogether*

Besides causing weakness or paralysis of movements pyramidal lesions produce increased muscular tone and exaggeration of the tendon reflexes

When the pyramidal system is suddenly damaged or destroyed as by hæmorrhage or injury there is a temporary depressant effect

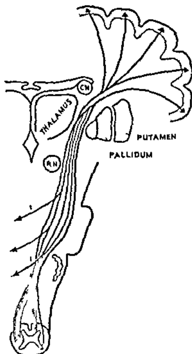


Fig 57—Diagram of the course of the pyramidal tract

on the lower motor neurones The resulting paralysis is accompanied by loss of muscle tone and abolition of tendon reflexes If the patient survives the characteristic hypertonia and increased reflexes of a pyramidal lesion appear after a time

(3) *The extra pyramidal system*—This is a term applied to the higher centres in the nervous system excluding the motor cortex and pyramidal tracts which are concerned with movement and

posture The system includes the basal ganglia the subthalamic nuclei the substantia nigra the red nuclei and other structures in the brain stem The connections of these extra pyramidal centres are imperfectly understood but they include fibres from the cerebral

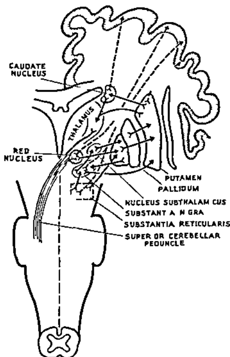


Fig 58 —To illustrate the chief afferent connections of the extra pyramidal system

cortex and the thalamus As far as is known there are no direct pathways from the basal ganglia to the spinal cord the connections with the lower motor neurones are indirect via several paths arising in the brain stem these include the rubro spinal reticulo spinal vestibulo-spinal and olivo spinal tracts (Figs 58 and 59)

Little detail is known of the functions of the extra pyramidal system It is concerned with the regulation of muscle tone and appears to play a part in the production of the more automatic bodily movements Diseases affecting the extra pyramidal system

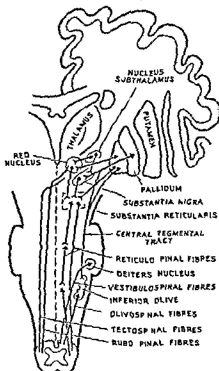


Fig 59 —To illustrate the chief efferent connections of the extra pyramidal system

are characterized by alterations in muscle tone and the appearance of involuntary movements Muscular power is rarely weakened but movement is often slowed

(4) *The cerebellum* —The cerebellum receives afferent fibres from the spinal cord vestibular system and cerebral cortex It influences the lower motor neurone mainly through its connections

via the thalamus with the basal ganglia and cerebral cortex It regulates the range rate rhythm and force of muscular contractions Lesions of the cerebellum cause muscular hypotonia and inco ordination (ataxia) No paralysis results although there may be fatiguability of muscles The inferior vermis is especially concerned with equilibration and disease affecting this part produces trunk ataxia Patients thus affected have difficulty in standing erect and tend to fall backwards or less frequently forwards

The sensory system —Sensory impulses from the periphery are conducted to the spinal cord by the afferent nerves through the posterior root ganglia and the posterior spinal roots These constitute the *first sensory neurones* But only a small proportion of these impulses ever reach consciousness as sensations The rest are concerned in the spinal reflex functions in the maintenance of the tone of the muscles or they terminate in the portions of the spinal cord or in higher centres such as the cerebellum which control the co ordination of muscular activity

Sensory impressions come not only from the skin and superficial tissues but also from muscles tendons and joints Disease of the first sensory neurones may consequently affect (1) cutaneous sensibility and abolish or disturb the perception and localization of tactile painful and thermal stimuli or (2) deep sensibility which is conveyed by the afferent fibres that come from the muscles tendons bones and joints This system underlies the recognition of position and movement and when cutaneous sensibility is lost heavy touches or the pain produced by pressure can be appreciated through it Owing to the wide anastomosis and distribution of the fibres of this system deep sensibility frequently escapes in areas in which cutaneous sensibility is lost then firm touches as those produced by a finger or the point of a pencil may be felt though light touches cannot be appreciated

After they have entered the spinal cord the various sensory impulse channels are rearranged and grouped into other systems The majority of the peripheral neurones that have carried them hither *terminate in the grey matter of the posterior horn at or near* the level at which they enter and from this grey matter the secondary sensory tracts take origin Some cross immediately or within a few segments to the opposite antero lateral column of the cord and in it ascend to the brain stem (Fig 60) Touch pain and

temperature are carried in this crossed secondary path. Other peripheral fibres however do not terminate in the grey matter of the spinal cord but run cerebralwards in the posterior column of the same side as that on which they entered the cord; these posterior column fibres carry the impulses upon which depend the appreciation of position of movement and of size and shape. Vibration sense is also conveyed in the posterior column and this contains too a path for touch. Consequently at any level of the spinal cord we have in each half of it two sensory paths conveying sensory impressions cerebralwards: one in the antero lateral column

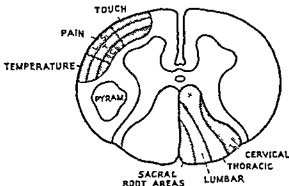


Fig 60.—To illustrate the position in a cross section of the spinal cord of the chief sensory paths

carries touch pain and temperature from the opposite half of the body and a second in the posterior column conveys the appreciation of posture weight size shape and other qualities of sensation from the same side of the body (Fig 60). A unilateral lesion of the spinal cord therefore produces the Brown Sequard phenomenon in which pain and thermal sensibility are lost below the level of the lesion on the opposite side of the body while on the side of the lesion there is in addition to spastic paralysis disturbance of the sense of position and of movement and loss of the recognition of weight size and shape and vibration. As touch has a double path one on the same and another on the opposite side of the spinal cord it is rarely much affected by unilateral spinal lesions.

At the upper end of the spinal cord the posterior-column fibres

terminate in the nuclei of Goll and Burdach and the impulses they carry are taken up by secondary sensory fibres which immediately cross to the opposite side of the medulla in the fillet decussation Consequently in the medulla oblongata all sensory impressions are

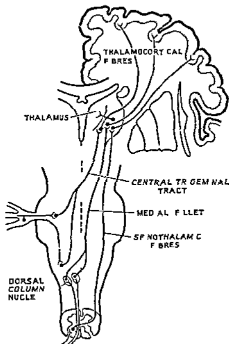


Fig 61 —Diagram of the chief sensory paths within the central nervous system

carried in secondary tracts which lie on that side of the nervous system opposite to the half of the body from which they come But even here all do not run in a single path for pain and thermal impressions pass through the lateral part of the bulb while those conducted by the posterior columns enter the medial fillet (Fig 61) Higher in the brain stem the two great sensory pathways are joined

by the secondary fibres from the nuclei of the sensory cranial nerves Finally the fibres of the fillet and spino thalamic tract terminate in the thalamus no secondary sensory fibres passing uninterrupted beyond it and from this a tertiary system of sensory fibres conveys sensory impressions by way of the internal capsule to the cerebral cortex (Fig 62)

The exact extent of the cerebral cortex concerned in reception of sensation is still doubtful it certainly lies mainly in the parietal

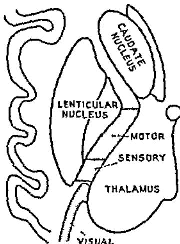


Fig 62 —The internal capsule of the left hemisphere showing positions occupied by the motor, sensory and visual projection fibres

lobes behind the fissures of Rolando It seems probable however that certain sensory qualities such as pain enter consciousness at a subcortical level in the thalamus The courses of the fibres and the position of the centres for the special senses are described in the section dealing with the cranial nerves (p 278) The speech centres and their connections are described at p 275

The spinal cord —The cord extends as far down as the interspace between the 12th thoracic and 1st lumbar spines the membranes are continued down as far as the body of the 2nd *sacral vertebra*

The cervical enlargement reaches to the 7th cervical spine Its largest part is opposite the disc between the 5th and 6th cervical vertebræ

The *lumbar s gments* lie opposite the 10th and 11th thoracic spines and the next interspinous space

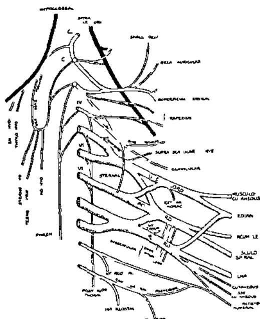


Fig 63 —Cervico-brachial plexus and its branches
(Aft P St w t Diag is f V us Di as)

Physiologically the cord is to be regarded as made up of a series of superimposed segments from each of which a pair of nerve roots arises To localize focal lesions of the cord it is necessary to be acquainted with the functions of each segment and therefore with the area of supply of the pair of nerve roots arising from it

Figs 63 and 64 show the distribution of the cervico brachial and

lumbo sacral plexuses The sensory functions of the cord are shown in Figs 65 and 66

Since the cord ends at the level of the lower border of the first lumbar vertebra spinal segments evidently do not correspond

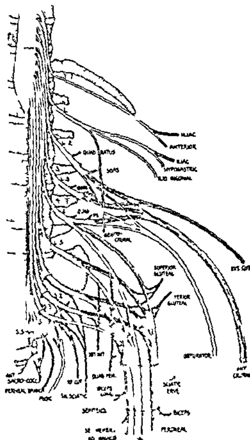


Fig 64 —Lumbo-sacral plexus and its branches
(A/I P rs Ste art Diagno l f Nervous Dis as s)

numerically with the vertebræ overlying them To determine which spinal segment is related to a given vertebra

For the cervical vertebræ add 1
For dorsal 1 to 6 add 2
For dorsal 7 to 9 add 3
The 10th dorsal arch overlies lumbar 1 and 2 segments
The 11th dorsal arch overlies lumbar 3 and 4 segments
The 12th dorsal arch overlies lumbar 5
The 1st lumbar arch overlies the sacral and coccygeal segments

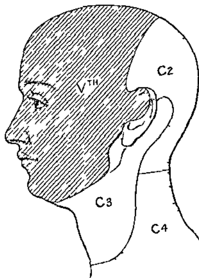


Fig 65 —Lateral view of the skin areas supplied by the Vth cranial nerve and the 2nd 3rd and 4th cervical segments

It is also necessary to remember that in the lower dorsal region the tip of a spinous process is on a level with the body of the vertebra below

Fig 66 (p 266) shows the sensory distribution of the posterior nerve roots (root areas)

CERVICAL SEGMENTS 5, 6, 7, 8 — DORSAL 1	
SHOULDER	SUPRASPINATUS
	TERES MIN
	DELTOID
	INFRASPINATUS
	SUBSCAPULARIS
	TERES MAJOR
ARM	BICEPS
	TRICEPS
FOREARM	BRACHIO RADIALIS
	SUPINATOR
	EXTENSOR CARPI RADIAL
	PRONATOR TERES
	FLEXOR CARPI RADIAL
	FLEXOR POLLIC LONG
	ABDUCT POLL LONG
	EXTENS POLL BREV
	EXTENS POLL LONG
	EXTENS DIGITOR
	EXTENS INDICIS
	EXTENS CARPI ULN
	EXTENS DIGITOR MIN
	FLEXOR DIGITOR SUBLIMIS
	FLEXOR DIGITOR PROFUND
	PRONATOR QUADRAT
	FLEX CARPI ULN
	PALMARIS LONG
HAND	ABDUCTOR POLL BREV
	FLEXOR POLL BREV
	OPPONENS POLL
	FLEXOR DIGIT MIN
	OPPONENS DIGIT MIN
	ADDUCT POLL
	PALMARIS BREV
	ABDUCTOR DIGIT MIN
	LUMBRICALES
	INTEROSSEI

Segmental innervation of muscles of the upper limb

D12 L1 L2 L3 L4 L5 S1 S2

HIP

ILIO PSOAS
TENSOR FASCIÆ
GLUTEUS MEDIUS
GLUTEUS MINIMUS
QUADRATUS FEMORIS
GLUTEUS MAXIMUS
OBTURATOR INTERN

THIGH

SARTORIUS
ADDUCT LONG
QUADRICEPS
GRACILIS
ADDUCTOR BREVIS
OBTURATOR EXT
ADDUCT MAGN
ADDUCT MINIM
SEMITENDINOSUS
SEMIMEMBRANOSUS
BICEPS FEMORIS

LEG

TIBIALIS ANT
EXTENS HALL LONG
EXTENS DIGIT LONG
SOLEUS
GASTROCNEMIUS
PERONEUS LONG
PERONEUS BREV
TIBIALIS POST
FLEXOR DIGITOR LONG
FLEXOR HALLUC LONG

FOOT

EXTENS HALL BREV
EXTENS DIGIT BREVIS
INTRINSIC MUSCLES OF THE FOOT
INTEROSSEI

Segmental innervation of muscles of the lower limb

Plate 21 shows the position of the different tracts of the cord on transverse section

The tables on pp 264–5 show the segmental innervation of the

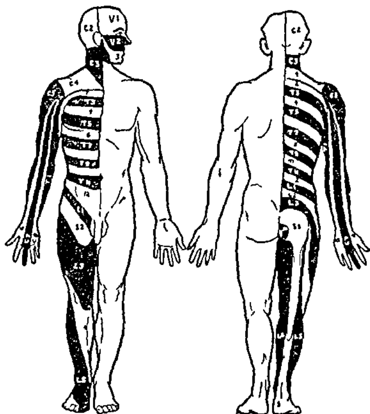


Fig. 66 —Diagram of cutaneous areas supplied by sensory roots Minor variations are common

(From Elliott Hughes and Turner "Clinical N[illegible] Cas[illegible])

muscles of the limbs It may be found convenient for reference in the study of cases of peripheral paralysis The nerve supply of the head is considered along with the cranial nerves (p 278)

The peripheral distribution of the chief sensory nerves is indicated in Figs 65–68

Vascular supply of the brain and spinal cord —The brain is supplied by the internal carotid and vertebral arteries Owing to the position of origin of the left common carotid an embolus can enter it more easily than it can the artery of the opposite side Embolic

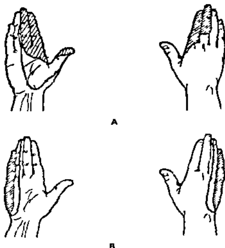


Fig 67

A A ea of cutane s sensory l ft r divisi n f th median n rve in th rm. A of l t l ght to h b d d by th co tin lin a d th t t pin pri k is indic ted by h bl q lines. B A of cut eo s se sory l s ft divis f th l erv bo the elbow Anæsth t rea (l ght t h) is limited by th contin lin d nalg c ar (pin-pri k) by th r ss-shading.

lesions are therefore more frequent in the left than in the right cerebral hemisphere

The two *vertebral arteries* unite at the lower border of the pons to form the *basilar* which runs up the middle of the anterior surface of the pons and ends by dividing into the two posterior cerebrals It gives off paramedian and short and long circumferential branches which supply the pons and parts of the mid brain and *cerebellum* *These not infrequently become thrombosed*

The *posterior cerebral* supplies the occipital lobe the lower part of the temporal lobe with the uncinate gyrus the inner part of the crus and the corpus quadrigeminus and the posterior part of the posterior limb of the internal capsule *Blocking of this artery at* its origin will therefore involve the visual centre and the sensory fibres but thrombosis often involves the calcarine branch and hence the visual centre alone

The *basilar artery supplies the upper surface of the cerebellum* the vertebral supplies its lower surface as well as the greater part of the medulla oblongata

The *internal carotid* gives off the *anterior cerebral* artery which *curves round the anterior end of the corpus callosum and is chiefly* distributed to the inner surface of the cerebral hemisphere as far back as the parieto-occipital fissure It also supplies the superior

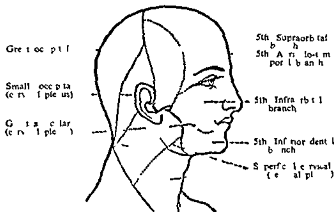


Fig 68 —Distribution of the sensory nerves of the head Compare with it the segmental distribution as shown in Fig 65

frontal convolution and gives a branch to the anterior part of the internal capsule and to the basal ganglia

The internal carotid is mostly continued on to the brain as the *middle cerebral* which lies in the Sylvian fissure An embolus which has found its way into the internal carotid therefore usually ends in the *middle cerebral or one of its branches* The middle

cerebral gives off *cortical branches* which supply the motor area and the upper part of the parietal and temporal lobes These branches anastomose freely with those of adjoining arteries hence blocking of one of them may be largely compensated by the establishment of a collateral circulation It also gives off *central branches* which penetrate into the brain substance and supply the white matter and the basal ganglia There are two chief groups of these central arteries—an anterior group called the *lenticulo striate* and a posterior group the *lenticulo optic* As the lenticulo striate are more directly exposed to the force of the wave of arterial blood they are more frequently ruptured than are the lenticulo-optic These central arteries do not anastomose with one another They are therefore to be regarded as end arteries Hence it is that a lesion of one of them is much less likely to be compensated than is a lesion of a cortical branch

The venous blood from the brain is poured into the *venous sinuses* Owing to the slow current in these thrombosis readily occurs The blood from the interior of the lateral ventricles is chiefly *returned by the veins of Galen* which end in the *straight sinus*

Spinal arteries —The *anterior and posterior spinal arteries* arise from the vertebrals and travel downwards in the pia mater the former in the antero median fissure and the two latter alongside the posterior nerve roots Although they have a long and tortuous course they do not diminish in size being reinforced by radicular tributaries from the intercostal and lumbar arteries The anterior spinal artery supplies most of the spinal cord only the posterior parts of the posterior horns and columns being supplied by the posterior spinal arteries

The chief veins of the spinal cord are situated dorsally and ventrally in the middle line Like the arteries they communicate by radicular branches with the lumbar and intercostal veins and empty into the vertebral veins The blood in them flows upwards hence in compression of the spinal cord as by tumour or tuberculous abscess there is venous engorgement below the level of pressure This results in asphyxia of the spinal cord with paralysis and loss of sensibility which frequently disappears if the cause of compression is removed It also leads to increase in the protein content of the cerebro spinal fluid below the site of compression the main feature of Froin s syndrome

The routine method of examining the nervous system is described in the following pages The mental state and intellectual functions including speech are investigated first The cranial nerves are tested next followed by the motor sensory and reflex functions of the limbs and trunk

INTELLECTUAL AND MENTAL FUNCTIONS

It is important to arrive at some idea of the patient s intellectual state early as it affords indications that are of help in the subsequent investigation of his symptoms For example if his memory is deficient only a limited value is attached to the account that he gives of the onset of his illness or the state of his previous health Or if he is comatose or unable to understand speech any attempt to investigate the state of his sensory functions is likely to be frustrated It may be added that it is often essential to obtain a history from the relatives or friends of patients suffering from neurological or psychiatric disorders

1 Appearance and behaviour —The patient s bearing or his actions when lying in bed are to be noted Mention whether the patient is unduly disturbed or apathetic or whether he is in a state of agitation or terror Is his attention easily held or fleeting ?

Is he well groomed or unkempt ? What is the condition of his hair his beard his nails and his hands ? Any other feature which strikes one as uncouth in his behaviour e g facial tics abstraction with his own thoughts should be noted Is there any dimming of consciousness stupor or coma ?

Note whether his conversation flows easily or not whether he is mute answers only by monosyllables or is over talkative Do his remarks hold together as replies to questions ? Do they show flight of ideas (a rushing stream of ideas with some connection) or the Knight s move in association (when one remark follows another with only indirect connection) ? Do his replies to questions suggest thought blockage or does he keep on repeating your question or his own remarks (perseveration) ? He may use strange words (neologisms) or normal words strung together oddly (word-salad)

2 Emotional state —It is important to note his mood Is he happy or distressed ? Is he happier than his condition would

warrant (elation) or filled with despair or dismay (depression)? Does his conversation lead you to feel that there is flattening of emotion e g he speaks of family or financial success without pleasure or in an incongruous manner (he laughs after relating a misfortune or breaks into tears when given some pleasant news)? Does the play of his features suggest that he enjoys a private world of his own such as smiling or grimacing at odd times? Does he appear perplexed? Ask him whether persons and things seem as real as they once were or whether they seem changed in some mysterious way (depersonalization) Note whether he seems irritable or resentful or whether he receives your words with suspicion

3 **Delusions and hallucinations** —Delusions are false beliefs which continue to be held despite evidence to the contrary Hallucinations are false impressions from the organs of special sense (aural visual olfactory etc) for which no cause is found The patient s conversation may have already indicated that these are present. Note their content and the patient s attitude when you express doubt about what seems so real to him

Neither the delusions nor the hallucinations may be voiced *spontaneously They have then to be inquired for and the* introduction of the subject may call for considerable diplomacy Is his mind or his body interfered with by others or by some physical agency electricity wireless atoms chemical agencies poison? Has he felt that others talk about him or shun him? Are his relatives neighbours colleagues at work kind to him or difficult to get on with? Has he ideas of supernatural power inordinate wealth or conversely does he feel that he is weakened physically morally or financially?

Hallucinatory experiences may be carefully hidden Aural and visual false sense impressions are commonest Does he hear or see anything unusual? Does he taste or smell what he has not expected? *Hallucinations may only occur at certain parts of the day or in certain places* and this should be noted Delusions which are secondary to the hallucinatory experiences must be noted e g that the neighbours are against him because his bedroom is filled with a gas which he assumes the neighbours have engineered Notes should be made of any unusual actions upon the patient s part which have been prompted by delusions or hallucinatory ideas e g ideas that one is in the pay of his enemies or that he

clutches at small animals which he sees crawling over his bed clothes

4 Orientation in place and time —Does the patient appreciate his surroundings know where he is or is he wholly ignorant and does he try to explain his ignorance in some way such as confabulation? Can he tell the date approximately if not perfectly correctly?

5 Clouding of consciousness The states of clouded consciousness are important to recognize *Coma* is a state in which the patient makes no psychologically understandable response to external stimulus or to inner need In *semi coma* the patient although inaccessible does show some response for instance to painful stimuli Above these deep levels of altered consciousness lie various degrees of confusion from severe to slight The questioner must be alert to observe any minor defects in the patient s capacity to grasp what is and what has happened Such defects will usually be manifest in the responses to tests for orientation recent memory and appreciation of environment

6 Memory —Inability to grasp and retain images and ideas is a marked feature in acute toxic delirious reactions and in the subacute and chronic organic psychotic reactions In these cases recent events may have been registered but cannot be recalled though it is probably more correct to say that they have not even been registered

The degree to which recent memory is lost is an index of the degree of organic brain disorder (not necessarily permanent) Inquire about the day of the week and of the month the name of the Monarch and of the Prime Minister Ask the patient to recall what he has read in the paper or heard on the wireless More incipient changes are discovered by seeing whether the patient can repeat seven digits forwards or five digits backwards Bring up a subject discussed three minutes previously and note how much is remembered

7 General intelligence —It is usually necessary to ascertain the patient s general intelligence or how it has been affected by brain injury or disease (e g encephalitis) The standard which he reached before leaving school and the character of his work and his work

record since give a rough and ready approximation Frequent changes of job may indicate mental defect or social defectiveness (inability to get on with colleagues) Frequent changes after an accident or a serious illness with a previously good work record is suggestive of mental impairment

Tests of memory as given above will indicate the more serious defects and these can be further exposed by tests of reasoning more particularly where the tests show inability to criticize Ask the patient to take sevens from a hundred or to reverse in his mind s eye the hands of a clock The absurdities test What would be absurd if I told you I had three brothers John Fred and myself etc indicates grosser disability A man with relatively low intelligence can give the months of the year parrot fashion but is unable to say which month precedes May and which October etc etc

Inquiries should be made about the patient s **sleep** Does he sleep too much or too little ? If too much is any particular action likely to precipitate the hypersomnia If too little is the difficulty that of falling off to sleep or of waking frequently or waking early in the morning and being unable to go to sleep again Where there is no physical cause for the insomnia such as pain cough or asthma or the wearing of some uncomfortable plaster the insomnia is likely to be due to some psychological disorder e g the restlessness of mania the early waking in endogenous depression (melancholia) or the turmoil of the mind with difficulty in getting off to sleep in the reactive depressions (anxiety states)

Inquire about **dreams** These are frequent and sleep disturbing in the anxiety states whether the source of the anxiety is known or unknown In these conditions the sleep is not refreshing and a positive complaint of being as tired in the morning as at night may be made

SPEECH

Proceed next to the investigation of the **speech functions** In considering speech it is essential to distinguish between defects of articulation and enunciation and those disturbances of speech due to diseases of its cerebral mechanism which we speak of as aphasia

Supposing that the patient is able to speak one should note

whether there is any peculiarity in his articulation The following are the chief abnormalities which may be present —

1 **Stammering** —This requires no special description

2 **Lalling or baby speech** —Ask the patient to read something aloud If he lalls one will recognize that all the difficult consonants are dropped he speaks like a baby and if a child may perhaps make use of words of his own invention—idioglossia Lalling and idioglossia are usually the result of a congenital defect in the *appreciation of the meaning of sounds—congenital auditory imperception*

3 **Scanning or staccato speech** —The patient speaks slowly and deliberately syllable by syllable as if scanning a line of poetry Ask him to say artillery he will pronounce it ar til ler y *This is the kind of speech found in some cases of disseminated sclerosis*

4 **Slurring speech** —The syllables are slurred together as in a state of intoxication Thus British Constitution becomes Brizh Conshishushon This kind of speech is met with typically in general paralysis of the insane

5 **Dysarthria** or disorder of articulation is due to paresis or incoordination of the peripheral mechanism of speech either of the larynx tongue or lips though often all three are affected together When it is severe (anarthria) sounds can no longer be emitted as is the case in advanced bulbar or pseudo bulbar paralysis

If the patient s defect consists not so much in a disturbance of articulation as in an ability to produce speech or to understand it when spoken or when written then his condition is described as one of **aphasia**

In order to understand the method of investigating a case of aphasia it must be remembered that for purposes of speech we have (1) a producing mechanism This consists of two parts—one concerned with the production of spoken speech the other in the production of written speech (2) A receiving mechanism This also consists of two parts—one for the reception of spoken speech *the other for the reception of written speech*

We may thus classify cases of aphasia as follows —

1 Lesions of productive mechanism (motor aphasia)	Motor aphasia (loss of power of talking) Agraphia (loss of power of writing)
2 Lesions of receptive mechanism (sensory aphasia)	Auditory (word deafness) Visual (word blindness)

It must be borne in mind however that it is the exception to meet with a case of aphasia of a pure type Thus a patient may have both motor aphasia and also word deafness he may be unable to read as well as unable to write and so on

The *cortical centres* for the production and reception of speech are situated in the left cerebral hemisphere in right handed persons in the right hemisphere in the case of those who are left handed Hence the importance of ascertaining early in the investigation of a nervous case whether the patient is right or left handed

The centre for spoken speech occupies the posterior extremity of the 3rd frontal convolution (Broca s convolution) and the lower end of the ascending frontal

The centre for the production of written speech is believed to be in the posterior end of the 2nd frontal convolution

The centre for the reception of spoken speech is in the posterior half of the superior temporo sphenoidal convolution and that for the reception of written speech (visual speech centre) extends from the posterior part of this convolution into the angular gyrus (*see* Plate 20 facing p 250)

For practical purposes it is best to proceed with the investigation of aphasia in this order —

I SPOKEN SPEECH

1 *How is it received and interpreted?*—First find out whether the patient s hearing is good If so ask him to put out his tongue shut his eyes etc If he does so test him as to his understanding of nouns by asking him to touch his nose ear chin, forehead etc in turn Then test his verbs by asking him to smile to whistle etc Finally put to him longer questions or give him more complicated orders because when the disturbance of speech is only slight he may be able to understand simple questions and commands but not more complicated ones If the patient responds satisfactorily

to these tests he has evidently no difficulty in interpreting the meaning of words heard—i e there is no *word deafness*

2 *How is it produced?*

i If the patient can use only a few words make a note of what these are If he repeats any word or phrase again and again (' recurring or repetitive utterance), note what it is

ii If he has a considerable vocabulary (*a*) make a note of any examples of lalling slurring etc as described at p 274 This affords an indication of his *power of articulation*

Test him with such words and phrases as British Constitution West Register Street Biblical criticism artillery

(*b*) Show him common objects—a knife a pen a matchbox etc —and ask him to name them or if he is dumb to indicate with his fingers the number of syllables in the name of each If unable to fulfil these tests he has evidently got some forgetfulness of words (amnestic aphasia) Sometimes the patient has a general idea of the word he wants to use but forgets exactly how to pronounce it he omits some syllables or substitutes others for them so that the listener may hardly be able to make out what word he wishes to use

(*c*) If he makes mistakes in his use of words calling the knife a pen or vice versa he is suffering from paraphasia In that case one should note whether or not the patient shows that he is aware of his error by trying to correct himself or whether he goes on talking gibberish

3 *How is it repeated or echoed?*—Ask him to repeat words after you If he is word-deaf try to make clear your request by the aid of pantomime repeating the word or phrase over and over again If he is able to repeat what you say endeavour to find out whether or not he understands what he is saying

II WRITTEN SPEECH

1 *How is it received or interpreted?*—Ascertain whether or not his sight is good If so write on a piece of paper such questions or commands as How old are you? Put out your tongue etc If he does not respond satisfactorily there is some word blindness present—i e the patient has visual aphasia Inability to read is called alexia

2 *How is it produced?*—Ask him to write his name (This can often be done when all other power of writing is lost) If he is able to do so ask him some simple question—e g How many do two and

two make ?—and get him to write a reply If he has word-deafness put your question in writing If his right hand is paralysed make him write or print with his left If he writes pretty well get him to write an account of his illness and note whether he makes use of the wrong word at times (paragraphia) or whether there is repeated use of any particular word

3 *Can he write to dictation or copy ?*—Try using some simple book *If he succeeds endeavour to ascertain whether or not he* understands the meaning of what he writes

III PHENOMENA ASSOCIATED WITH SPEECH

1 *Does he und rstand pantomime ?*—Does he nod his head for yes shake it for no and can he indicate numbers with his fingers ? Loss of gesture language is termed **amimia** Mistakes in the use of gestures—e g nodding for no or shaking the head for yes —are termed **paramimia**

2 *Does he understand symbols*—e g numerals ? One may write down—

2	2	2
2	2	2
–	–	–
4	5	6

and ask him to point out which is right Inability to understand and manipulate mathematical symbols termed **acalculia** occurs in posterior parietal lesions affecting the dominant hemisphere If he can read music test him with musical notes

3 *Can he recogni e common objects ?*—Place beside him a pencil a coin and a match Ask him to strike a light, or to write something down If he is unable to select the proper article for the purpose he is suffering from **mind blindness** or **visual agnosia** provided of course that his visual mechanism is intact Inability to recognize his friends is another proof of the same condition

It occasionally happens that a patient who has neither motor nor sensory paralysis nor ataxia cannot perform certain acts though he can easily execute their component movements He is con sequently unable to make use of objects though he can recognize their use This condition is known as **apraxia** It results from destruction of the left hemisphere or of its connections through the corpus callosum with the right hemisphere It affects only

the left limbs i e the right hemisphere when the callosal fibres only are injured but it is usually bilateral It may be tested for by asking the patient to use certain objects or make or imitate certain movements For instance he may be given a box of matches and a cigarette and asked to light the latter If there is apraxia he may fail to open the box or to take a match from it or to strike the match or even to light the cigarette with the match if he has succeeded in striking it It is of course important to make sure that the patient understands the order

CRANIAL NERVE FUNCTIONS

In this section we propose to give a brief resumé of the essential points in the anatomy of each cranial nerve to indicate its functions and in some cases the chief symptoms which result from its paralysis and then to describe the method in which one investigates the state of the nerve at the bedside

FIRST OR OLFACTORY NERVE

Anatomy—The central processes of the bipolar sensory cells in the olfactory epithelium pass through the cribriform plate to the olfactory bulb where the cells of the second olfactory neurones lie Nerve fibres pass thence to the olfactory area of the cerebral cortex the piriform lobe

Test—Have three small bottles containing some oil of cloves some oil of peppermint and some tincture of asafœtida Apply these to each nostril separately and ask the patient if he recognizes them In testing avoid the use of such irritating substances as ammonia for these act partially through the 5th nerve The sense of smell may be abolished This is known as anosmia Before concluding that the nerve is at fault take care to exclude local changes in the nose itself—e g catarrh Parosmia is the name applied to that condition in which the sense of smell is perverted so that for instance offensive substances seem to have a pleasant odour and vice versa

Inquire also regarding hallucinations of smell These sometimes constitute the aura of an epileptic fit

SECOND OR OPTIC NERVE

Anatomy—From the retina which is the end-organ of the sense of sight the fibres of the optic nerve pass back to the optic chiasma Here

the fibres from the inner half of each retina decussate whilst those from the outer half remain on the same side Each optic tract therefore consists of fibres from the outer half of the retina on the same side and the inner half of the retina on the opposite side Each tract passes back to the superior corpus quadrigeminum and to the lateral geniculate body and the pulvinar of the thalamus of the same side In these which are known as the primary optic centres most of the fibres of the optic tracts terminate But another system of fibres which is known as the optic radiation, takes origin in the lateral geniculate body and passes through the posterior limb of the internal capsule and then backwards to the cortex around the calcarine fissure This therefore constitutes the chief visual centre and represents the opposite half of the field of vision the left half of the field of vision being represented in the cortex of the right hemisphere and vice versa (Plate 24)

Test —In testing the optic nerve one has to investigate three functions (1) Acuity of vision (2) field of vision (3) colour sense

Certain preliminaries must always be conducted One of these is to see that any error of refraction in the patient s eye is first corrected and that also there is no opacity of his media another is to take care to examine each eye separately

1 Acuity of vision —If this is very much diminished it may be doubtful whether the patient can tell light from darkness To investigate this place the patient in a darkened room opposite to a lamp and alternately cover and uncover his eye What is perhaps a better plan is to concentrate the light upon his eye by means of a mirror or lens and ask him to say when it is light and when it is dark

In lesser degrees of impairment, ask the patient to count fingers This is done by placing him with his back to the light while the observer standing face to the patient holds up a varying number of fingers of one hand and asks the patient to say how many there are The test should be applied at different distances

For the detection of slight degrees of impairment of visual acuity Snellen s types will be found useful These consist of letters of different sizes each of which should be capable of being read at a definite distance—the largest at 60 metres the smallest at 6 In using the types the patient is placed with his back to the light while the types are placed level with the eye at a distance of 6 metres (about 20 ft.) He is then asked to read the letters from

above downwards For the purpose of recording the result the following symbols are employed —

V = visual acuity
d = distance of eye from type (i e 6 metres)
D = distance at which type should be capable of being read

Suppose that at 6 metres the patient is able to read the smallest type—that is to say that which should be readable at 6 metres off Then his visual acuity (V) = $\frac{d\text{ (i e 6 metres)}}{D\text{ (i e 6 metres)}}$ or normal

But if at 6 metres he can only read the size of type which one should be able to read at 60 metres V = $\frac{6}{60}$

The term amblyopia (literally blunt-eyedness) is often used to mean defective vision The term amaurosis (literally dark ness) is sometimes used to signify complete blindness

2 **Field of vision** —When we fix the eye upon an object we not only see that object but also a number of objects in the neighbour hood more or less distinctly The sum of the objects that form images upon the retina whilst the eye is gazing in one particular direction is called the field of vision It should be noted that although the field of vision differs with each different act of fixation the peripheral limits are the same and are largely determined by the margins of the orbit nose and cheek A rough estimate of the extent of the field of vision for large objects may be obtained in the following way —

Seat yourself opposite to the patient and at a distance of about half a yard from him If his right eye is to be tested ask him to place his hand upon his left and to look steadily at your own *left* eye Look steadily yourself at the patient s right eye your own right being closed and hold up your left hand in a plane midway between his face and your own and at first at almost full arm s length off Keep moving the fingers of the hand and bring it nearer until you can just yourself with the tail of your eye catch the movement of the fingers Then ask the patient whether he sees them telling him meanwhile to be sure not to take his own eye off yours If he fails to see the fingers keep bringing the hand nearer until he does see them Test the field in this fashion in every

direction—upwards downwards to right and to left—using the extent of your own field for purpose of comparison

This gives the outline of his field for appreciation of a moving object which may however be relatively intact when the fields for other forms of stimulation are seriously constricted In consequence his field for appreciation of a stationary object must also

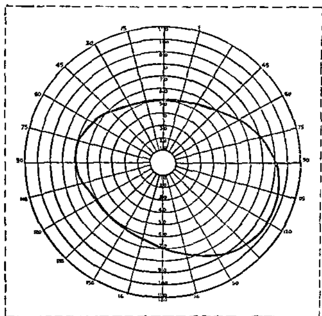
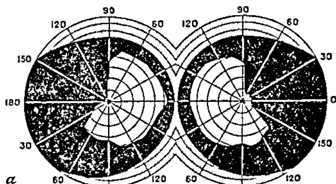
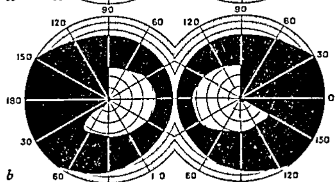


Fig 69 —Extent of the right field of vision with a white target 20 mm in diameter mapped on a perimeter at a distance of 33 cm

be investigated in a similar manner but by asking the patient to indicate when he sees the observer s fingers held at rest

Considering the field of vision in more detail we appreciate that whereas the objects which cause images to fall upon the central part of the retina (the macula) are seen in minute detail and bright colouring objects further and further from the point of fixation are seen with less and less distinction and colour until


90
120
60
150
180
30
60
90
120
a
90
120
60
30
0
150
60
120
90


120
60
150
180
30
60
1 0
b
90
120
60
30
0
150
60
120

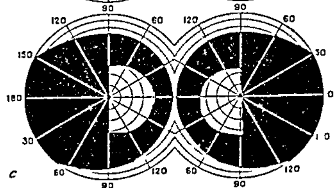

90
120
60
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180
30
60
90
120
c
90
120
60
30
0
1 0
60
120
90

Fig 70 —Fields of vision in a case of pituitary tumour (probably chromophobe adenoma) showing the development of a bitemporal hemianopia

a May 1947
V A Lt 6/12 Rt 6/18
b August 1947
V A Lt 6/12 Rt 6/12
c October 1947
V A Lt 6/12 Rt 6/12

The fields were plotted on a Bjerrum s screen at a distance of 2 000 mm from the eye to the point of fixation at the centre of the screen using a 10 mm white object The outline of the black areas in the charts is that of the average normal field of vision and the white areas show the patient s fields

(Courtesy of Dr W C Northfield)

at the periphery of the field we can only appreciate the presence of an object of considerable size without being able to judge its form To the temporal side of the central point of vision is the blind spot which represents the optic nerve head area in which there are no light receptors Perimetry surveys the field of vision and the limits of perception are charted The centre point of the chart corresponds to the visual axis the point of fixation is therefore the point of more distinct vision Around this point are arranged a series of more or less concentric lines each of which denotes equal visual acuity and is called an *isopter* For purposes of investigation we divide the visual field into three parts —

(1) An area surrounding the point of fixation to 20
(2) An intermediate area between 20 and 50
(3) An outer area from 50 to the periphery

The fixation point is not exactly central so that the outer and inner part of the field is unequally divided Further the boundary is delimited inwards and upwards by the nose and brow Testing with a large object we find the outermost limit of the field of vision reaches 104 outwards 50 upwards 70 inwards and upwards whilst down and in (owing to the obstruction of the most prominent part of the nose) it reaches from 45° to 50 (Fig 69)

The binocular field extends 200 or more laterally and about 140 vertically in the middle of which is a circular portion common to each eye with a diameter of about 120

On each side of this paired area is a semilunar area which is unpaired and which accounts for the remainder of the field

Perimetry is concerned with an investigation of the uniocular field of vision We have noted that the acuity of perception is very much lower at the periphery and gradually increases as we pass to the point of fixation We may test this acuity with objects of different size and we shall find that whereas a very small object is visible at and near the fixation point it fades from view as it is withdrawn towards the periphery By using a graduated series of objects we are able to plot out a series of isopters each of which corresponds to a known size of object used at a known distance from the eye

That part of the field between the periphery and the 30 circle is investigated by means of a perimeter —

A 3 mm white test object is usually used but when visual acuity is

impaired a larger diameter may be required Thus the fraction of diameter of object/distance $\left(\text{usually } \frac{3 \text{ mm}}{330 \text{ mm}}\right)$ is a measure of the visual acuity at the particular point on the field which is being tested

The area within the 30 circle is examined by means of test objects *upon a black screen—Bjerrum s screen—at a distance from* the patient of 1 000 to 2 000 mm

The patient is seated comfortably at this distance with the head steadied by a chin and head rest and a grey object 1 cm in diameter with a black centre is fixed to the screen on a level with the patient s eye

The blind spot is first of all mapped out with a white object 20–30 mm in diameter The peripheral field is next mapped out with a 1 mm. object and at a distance of 2 000 mm it should be circular and extend to about 26 that is to the edge of the 2 metre square screen With the small object areas of blindness or defective perception should be sought around the blind spot especially between this area and the macula the centrocæcal area and in the horizontal meridian on the nasal side of the fixation spot The findings are marked upon the screen with black pins and subsequently transferred to a chart

Changes in the field of vision —It may be contracted all round its periphery This is spoken of as **concentric diminution** of the field of vision It occurs in hysteria papillœdema some forms of optic atrophy bilateral lesions of the anterior part of the cortical visual centres and various affections of the retina

Sometimes the loss of vision is confined to the centre of the field. This is spoken of as a **central scotoma** Sometimes it is due to local disease of the choroid or of the retina in the neighbourhood of the macula In that case it may affect only one eye A unilateral central scotoma is also produced by optic or retrobulbar neuritis which in most cases is a symptom of disseminated sclerosis It is sometimes due to toxic causes or vitamin deficiency when it is generally bilateral Pressure on the optic nerve is another cause It may also result from a lesion of the posterior part of the cortical visual centres and is then bilateral but this is rare

The term **hemianopia** means loss of sight in one half of a visual field When the same half of both fields of vision is lost, the hemianopia is described as homonymous e g right homonymous hemianopia when the blindness occupies the right half of both the right and left fields (Plate 22 and Fig 70)

Superior and *inferior hemianopia* means loss of the upper and lower halves of the visual field respectively They are of rarer occurrence than the lateral variety and are sometimes spoken of as altitudinal hemianopia Hemianopia limited to one quadrant of the field is described as quadrantic hemianopia or quadrantanopia

Bitemporal hemianopia means loss of vision in the temporal or outer halves of both fields and is due therefore to loss of visual power in the nasal half of each retina It can only be produced by a lesion of the optic chiasma involving those fibres of the optic nerves which decussate and is accordingly rare It is commonly due to a tumour of the pituitary body but may be produced by inflammatory or traumatic lesions of the optic chiasma

Binasal hemianopia signifies a loss of the nasal or inner half of each field and indicates a diminution of visual power in the temporal half of each retina It can only be produced by a bilateral lesion confined to the uncrossed optic fibres on each side of the chiasma It occurs with excessive rarity

Temporal and nasal hemianopia are sometimes spoken of as *heteronymous* in contradistinction to the *homonymous* variety

3 Colour sense —This is tested by means of Holmgren s wools Throw all the skeins together on a table in good daylight keeping the test skein separate Explain to the patient that he is to match the colour not to name it and that he is to select all those skeins which are *like* it whether they are of a darker or lighter shade or not Show him first a pure pale green skein and ask him to match it If he does so correctly his colour vision is normal If on the other hand he selects one of the confusion colours (grey straw colour etc) he is to be regarded as colour blind

Total colour blindness is rare Red green blindness is the commonest form Yellow blue blindness is not nearly so common If the patient is totally colour blind he confuses with the test skein all those of equal brightness no matter what their tint may be If red green blindness is suspected show him a purple skein and he will select blue as a match for it—indicating that he fails to see the red element in the purple If he is blue blind he will select red or orange

Colour field —The field for colour is investigated in the same manner as is the field for white *For the same size of object the fields for*

VISUAL FIELDS

VISUAL PATHS

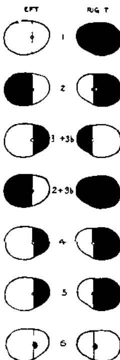


Plate 22 —The Visual Pathways

I t l p l co pl t bl nd right y w th l ss f d t ght fl p od b t mpo l h man pi

3 + 3b p d b l h mi p

+ 3b prod ce bl d ess f right with t mporal hem yn p f l ft vi l fi ld

4 prod ri ht h mon m h m p th h m an yn p pill ry po se

p od right h monym h p th rm l pup il rv t t l ght

6 prod es ght h mo yn t l h se t

R produc d f m D gno f Nervou D (S J mc P rce lcu b rt f Ed d A ld & C

Functions —The 6th nerve supplies the external rectus the 4th supplies the superior oblique All the other ocular muscles along with the sphincter pupillæ the muscle of accommodation and the levator palpebræ superioris are supplied by the 3rd

Ocular movements —Horizontal movement outwards is described as abduction inwards as adduction vertical movement upwards as elevation and downwards as depression The eye is also capable of diagonal movements at any intermediate angle Rotatory movements the eye rolling like a wheel towards the nose (internal rotation) or away from the nose (external rotation) do not occur

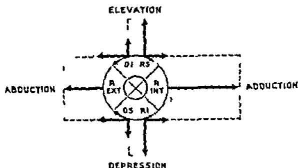


Fig 71 —Scheme to illustrate the action of the ocular muscles
(R Ext —e t rn l rectu R I t —internal ect O I —nf r or blig R S —perior ectus O S —supe or obl q e R I —infer or rectus) (*After Ma q r f m F chs*)

normally but may be seen in some varieties of ocular palsy Fig 71 shows the actions of the ocular muscles diagrammatically the length of the arrows indicating the relative power of each muscle in different directions It shows that the internal and external recti act directly in a single plane but all movements are normally the result of the concerted action of two or more muscles Thus when the eye looks straight forward elevation is the result of the combined action of the superior rectus and the inferior oblique depression of the inferior rectus and superior oblique In adduction the internal rectus is aided by the superior and inferior recti and in abduction the external rectus is aided by the obliques It will be seen that when two muscles act together in this way their

conflicting actions cancel out so that an orderly movement results

Normally the movements of the two eyes are symmetrical so that the visual axes meet at the point at which the eyes are looking This is spoken of as conjugate movement of the eyes Infranuclear (lower motor neurone) lesions of the third fourth and sixth nerves lead to paralysis of individual eye muscles or groups of muscles Supranuclear lesions lead to paralysis of conjugate movements of the eyes

Symptoms of paralysis *Sixth nerve* —Inability to move the eye outwards and diplopia on looking in that direction Possibly internal squint In nuclear lesions there is also loss of the power of conjugate deviation of both eyes horizontally to the side of the lesion

Fourth nerve —Impaired power of downward movement and on the attempt to look downwards the eyeball is rotated inwards by the inferior rectus Diplopia only below the horizontal plane with the images uncrossed but the false one tilted There is rarely a visible squint.

Third nerve —Ptosis the eye can only be moved outwards and a little downwards and outwards pupil usually dilated and fixed loss of power of accommodation

Paralyses of the 3rd nerve are not infrequently partial—only one or a few of these functions being lost

In order to estimate the degree of ptosis one must eliminate the action of the occipito-frontalis This is done by pushing down upon the latter muscle so that the eyebrows are kept level and then asking the patient to look up The extent to which the lids are raised indicates the strength of the levator It must be remembered that smooth muscle in the upper lid is innervated by the cervical sympathetic and exerts a tonic elevating action Slight ptosis therefore occurs after a lesion of the cervical sympathetic (*see* p 311)

Any retraction of the upper lid from over action of the levator should be noted by observing the relation of the edge of the lid to the upper margin of the cornea when the patient is looking straight forward

How to test these nerves —Thus the signs of a lesion involving any of these nerves may be—1 defective power of movement of the eye 2 the presence of a squint 3 the presence of diplopia Of these signs the last is really the most trustworthy of all for paraly-

of the muscle(s) supplied by the nerve may be so slight as to lead to no appreciable squint and to no visible defect in mobility

Strabismus

By *squint* or *strabismus* is meant a condition in which the visual axes do not meet at the point of regard Of this there are two varieties *paralytic* and *concomitant* and it is necessary that the two be carefully distinguished

Paralytic strabismus —The following are the characters of a paralytic squint —

(1) *Limitation of movement* —Paralytic strabismus is due to loss of power in one or more of the extraocular muscles a prominent feature therefore is lack of ability to move the eye in the direction of the physiological action of the muscle affected Although this lack of power is usually apparent sometimes the loss of muscular power is so slight or the unaffected muscles mask the loss of action of the affected muscle so much that the defective movement of the eye is hardly visible

Movements in the so-called *cardinal directions* are tested by fixing the patient s chin with one hand and moving the forefinger of the other in the direction indicated The eyes move normally 50 outwards 50 inwards 33 upwards and 50 downwards

If an eye fails to move at all or fails to move throughout the normal angular excursion the deviation of the eye in a direction opposite to the physiological action of the muscle is called the *primary deviation or squint* and it is measured by the angle which a line from the object to the nodal point of the eye makes with the visual axis (Fig 72a) If now we cover the unaffected eye and so cause the patient to take on fixation with the affected eye we shall find that the eye that is covered will deviate still more than the primary deviation of the affected eye (Fig 72b) This deviation of the healthy eye is the so-called *secondary deviation or squint* and this difference in amount between the primary and secondary deviation is the most important distinguishing feature between paralytic and concomitant strabismus

(2) *False orientation of the field of vision* —This is an erroneous judgment by the patient of the position of an object in that portion of the field of vision towards which the paralysed muscle should normally move the eye Take the case of a patient who has

paralysis of the right external rectus muscle If such a patient closes the left eye and is asked to touch suddenly an object held in the horizontal direction on his right side he will fail and will strike wide of the object on his right hand side

The explanation of the phenomenon of secondary deviation being greater than primary is that when fixation is assumed by the paralysed eye the same amount of nervous energy passes to the associated muscles

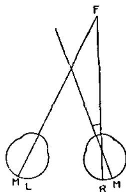


Fig 72a—Diagram to illustrate primary deviation in a case of paralysis of the right external rectus muscle

W th fi t b th l ft ey th d vi t of th ght y is ind t d. F = po t of fix t o M = m l

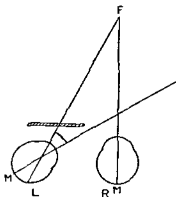


Fig 72b —Diagram to illustrate *secondary* deviation in a case of paralysis of the right external rectus muscle

W th fi tio by th right y th l ft eye wh n red by scree d viates inw d f m th d d th right y d ring fi t on by th l ft y (F g 72a)

of the two eyes in this example to the right external rectus and left internal rectus As however the right external rectus is paralysed an unusually great impulse is needed to stimulate it the result is that its associated muscle in the left eye is over stimulated and its movement too great

This same reasoning may be applied to explain the phenomenon of false orientation in that an object is projected into space largely according to the amount of nervous energy expended in moving the eye so as to fix that object and as in the case of paralysis the amount of nervous energy expended is in excess of movement the object is projected too far in the direction of the physiological action of the paralysed muscle

(3) *Vertigo* is occasionally a symptom of paralytic strabismus when both eyes are opened It is due partly to the confusion of double sight and partly to false orientation

(4) In order to overcome double vision the patient *turns the head in the direction of the action of the paralysed muscle* Information may therefore be obtained as to which muscle is involved by noting the way in which the head is held

(5) *Diplopia* —Patients with paralytic squint complain of double vision which is due to the fact that owing to the lack of movement of one eye in a particular direction the images of external objects do not fall upon identical points of the two retinæ thus double vision or *diplopia* is therefore in that part of the field of vision into which the paralysed muscle should move the eye were it unaffected

In health when fixing an object the image formed in each eye falls upon the macula so that not only are the two images of equal intensity and definition but since they fall upon identical points of the two retinæ they produce but a single image In paralytic strabismus the image of the object fixed falls upon the macula of the healthy eye and is seen with distinctness and detail and is called the *true image* whereas in the affected eye the image falls upon the retina outside the macula and as in consequence it is indistinct and blurred it is called the *false image*

The investigation of a case of paralytic strabismus and the diagnostic value of diplopia —First of all make certain that the diplopia is *binocular* since certain conditions astigmatism lens opacities etc may produce *monocular* diplopia

Movements in *cardinal* directions have already been mentioned and they consist of movements up down in and out The main action of each individual muscle is in a cardinal direction and so they are spoken of as lateral rotators elevators or depressors

All the rectus muscles arise around the apex of the orbit and pass forwards to be inserted into the sclera a varying distance behind the cornea With the eyes in the primitive position the external and internal recti turn the eyes to right and left only Owing to the direction in which the superior and inferior recti pass to be inserted although they act mainly as elevators and depressors of the eye they have a second component which causes them to act also as adductors

The superior and inferior oblique muscles also act mainly as depressors and elevators respectively but also act in a subsidiary way as abductors

By turning the eye outwards 27 the superior and inferior recti may be

made into almost pure elevators and depressors Similarly by turning the eye inwards the superior and inferior oblique muscles may be made into almost pure elevators and depressors

In this way we are able to resolve diplopia into horizontal and vertical (which much simplifies our investigation) and find the field of maximum diplopia in one of the cardinal directions

When the images in diplopia are separated laterally so that the right image belongs to the right eye and the left to the left eye the condition is spoken of as *homonymous or uncrossed diplopia* If however the left image belongs to the right eye and the right to the left it is called *heteronymous or crossed diplopia* It will also be found that the real image belongs to the healthy eye whereas the false image belongs to the paralysed eye

The production of homonymous diplopia —If as the result of paralysis of an abductor muscle (external rectus muscle) there is deviation of the eye inwards (convergent strabismus) the image in this eye will fall upon a point of the retina internal to the macula Two things will result (1) the image will not be so sharp as the image in the healthy eye proving that the *false* image belongs to the affected eye (2) since images that fall upon the retina on the nasal side of the macula are projected in space to the temporal side of the eye it follows that *paralysis of an abductor producing convergent strabismus causes homonymous diplopia* and also that the false image is projected in the direction of the physiological action of the paralysed muscle (Fig 73)

The production of heteronymous or crossed diplopia —If as the result of paralysis of an adductor muscle (internal rectus) there is a deviation of the eye outwards (divergent strabismus) the image in this eye will fall upon a point of the retina external to the macula As seen on p 292 the false image is produced in the affected eye and since images that fall upon the retina to the temporal side of the macula are projected into space to the nasal side of the eye it follows that *paralysis of an adductor producing divergent strabismus causes heteronymous or crossed diplopia* (Fig 74)

In a similar way it may be shown that in paralysis of an elevator muscle the false image (which belongs to the affected eye) lies on a higher level than the true image and in paralysis of a depressor muscle the false image lies below the true image (Figs 75 76)

If a rotator muscle is weak the false image is tilted

Actions of the muscles —The external and internal recti muscles move the eyes in a horizontal direction consequently maximum diplopia is produced when the eyes turn horizontally in the direction towards which the paralysed muscle normally turns the eye

We have seen that the superior and inferior recti muscles become simple elevators and depressors when the eyes are turned outwards

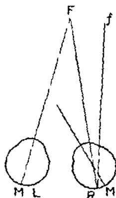


Fig 73 —In the case of paralysis of an *abductor* muscle (here the right external rectus) the result is *homonymous* diplopia

F is the point of fixation which produces an image on M the macula of the left eye the true image. As the image falls internal to the macula M in the right eye it is projected to f the false image Thus the false image belongs to the affected eye and projected in the direction of the physiological action of the paralysed muscle.

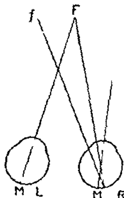


Fig 74 —In the case of paralysis of an *adductor* muscle (here the right internal rectus muscle) the result is *heteronymous* or *crossed* diplopia

F is the point of fixation which produces an image on M the macula of the left eye, the true image. As the image falls external to the macula M in the right eye it is projected to f the false image. Thus the false image belongs to the affected eye and is projected in the direction of the physiological action of the paralysed muscle

consequently maximum vertical diplopia is produced when the eyes look up and out and down and out respectively

The inferior and superior oblique muscles become simple elevators and depressors when the eyes are turned strongly inwards consequently maximum vertical diplopia is produced when the eyes look up and in and down and in respectively

In conjugate movements the muscles of the two eyes act in pairs (for instance in conjugate movements in the horizontal direction to the right the right external rectus muscle is linked in action

with the left internal rectus muscle) and the table below shows a series of six pairs each pair representing true associates

1 *Muscles moving the eyes laterally*
   (*a*) To the right
      Right external rectus
      Left internal rectus
   (*b*) To the left
      Left external rectus
      Right internal rectus

2 *Muscles moving the eyes upwards*
   (*a*) With the eyes turned to the right
      Right superior rectus
      Left inferior oblique
   (*b*) With the eyes turned to the left
      Left superior rectus
      Right inferior oblique

3 *Muscles moving the eyes downwards*
   (*a*) With the eyes turned to the right
      Right inferior rectus
      Left superior oblique
   (*b*) With the eyes turned to the left
      Left inferior rectus
      Right superior oblique

4 *Muscles which rotate the eyes*
   (*a*) External rotators
      Inferior rectus
      Inferior oblique
   (*b*) Internal rotators
      Superior rectus
      Superior oblique

Method of finding the direction of maximum diplopia —The patient is seated with the head fixed in position (preferably in a head rest) with a red glass before the right eye and a green glass before the left At a distance of about fifteen feet the observer moves a light in the direction indicated in the sub joined diagram each of the lateral squares corresponding to a pair of true associated muscles thus a maximum vertical diplopia produced when the patient looks

up and to the right into the right superior square shows that either the right superior rectus or the left inferior oblique muscle is the

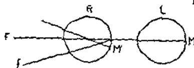


Fig 75 —In the case of paralysis of a *depressor of the right eye* it will be seen that the object F causes an image to fall above M the macula of the right eye the result is that the false image is projected to *f* below F the true image Thus the *lower* image belongs to the affected eye

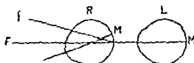


Fig 76 —In the case of paralysis of an *elevator* of the right eye it will be seen that the object F causes an image to fall below M the *macula of the right eye* the *result is* that the false image is projected to *f* above F the true image Thus the *higher* image belongs to the affected eye

L — The patient looks — **R**

Upwards to the left LEFT SUPERIOR AREA	Upwards SUPERIOR MEDIAN AREA	Upwards to the right RIGHT SUPERIOR AREA
To the left LEFT EXTERNAL AREA	Straight ahead PRIMARY AREA	To the right RIGHT EXTERNAL AREA
Downwards to the left LEFT INFERIOR AREA	Downwards INFERIOR MEDIAN AREA	Downwards to the right RIGHT INFERIOR AREA

Table for charting the field of diplopia in a case of paralytic strabismus

one affected It is only necessary to find to which eye the false image belongs to decide which muscle is paralysed The higher of the two images indicates the affected eye

Note that in using this chart the area of greatest vertical diplopia is the namesake either of the affected muscle or its true associate in the other eye further that the true associates always bear names which are the most contrary possible thus *left inferior oblique* is in every term opposite to *right superior rectus*

Concomitant strabismus —It has been explained when dealing with paralytic squint that the amount of angular deviation of the two visual axes varies with different positions of the two eyes and also that secondary deviation is always greater than primary deviation

In concomitant strabismus as its name implies the angular deviation of the visual axes is the same in whatever position the eyes may be in other words the primary and secondary deviation are always equal

As there are certain conditions which simulate squint it is important to make certain that the case under investigation is truly one of squint

Screen test —Steadying the patient s head by holding the chin with one hand ask him to look at an object immediately in front of him Suddenly cover the apparently fixing eye and ask the patient to fix the object with the uncovered eye If this eye makes any movement in taking up fixation it must have been previously deviating If now the eye behind the screen (which was previously fixing) be observed it will be seen to deviate in the same relative direction as was the other eye and *to the same angular amount* that is the primary and secondary deviation are equal

The clinical features of concomitant strabismus are —

(1) It always begins in early childhood over 70 per cent before the 5th year and the great majority before three years of age
(2) The movements of the eyes are good in all directions
(3) Diplopia is practically never a symptom
(4) The primary and secondary deviation are equal
(5) The deviating eye often has defective vision

A squint may be *periodic* or *constant* and if constant *monocular* when the same eye deviates whilst the other usually fixes or *alternating* when either eye fixes indifferently



Nystagmus —The term nystagmus is applied to a disturbance of ocular posture characterized by involuntary rhythmical oscilla tions of the eyes These movements may be horizontal vertical or rotary The speed of the movements may be the same in both directions or quicker in one direction than another in the latter case the quicker movement indicates the direction of the nystagmus To examine for nystagmus ask the patient to look straight in front of him and observe whether the eyes remain steady Then ask him to look to his extreme right then to the left and then upwards and downwards Observe the rate amplitude and rhythm of the nystagmoid movements in each direction and whether they are influenced by the position of the head (positional nystagmus)

A few irregular jerks of the eyes are often seen in full lateral deviation The brief duration and irregularity of these movements distinguish them from true nystagmus

Nystagmus is most commonly due to visual disorders affections of the vestibular system (either centrally or peripherally) lesions affecting the central pathways concerned in ocular posture or weakness of the ocular muscles Nystagmus of visual origin is pendular and often rotary on central fixation of the eyes *Con genital nystagmus sometimes occurs without discernible cau e and* it also shows this pendular quality

Conjugate ocular palsies —In addition to the defects of movement due to paralysis of the individual ocular muscles weakness or paralysis of the movement of both eyes in one direction sometimes occurs Thus the patient may be unable to look to either side or upwards or downwards or the power of convergence alone may be lost Weakness of conjugate lateral movement may occur in hemiplegia due to cerebral lesions especially in the acute stage Palsy of this movement occurs with lesions in the neighbourhood of the 6th nucleus of the side to which the movement is weak Bilateral paralysis of lateral conjugate movement is seen in centrally placed pontine lesions above the level of the 6th nerve nuclei The conjugate vertical palsies are always associated with disease of the corpora quadrigemina or in the neighbourhood of the oculomotor nuclei

If both eyes are kept persistently turned in one direction the condition is spoken of as **conjugate deviation of the eyes** It is usually either to the right or to the left Conjugate deviation of

the eyes may be brought about either by a lesion which produces paralysis or by one which causes irritation or spasm In the former case the eyes (and usually also the head) are turned towards the side of the lesion provided the latter is in the cerebral hemisphere The patient in fact is said to look towards his lesion An irritative lesion in a similar situation causes the deviation to be towards the healthy side If however the lesion has its seat in the pons these rules are just reversed the deviation being towards the sound side in a paralytic lesion and towards the affected side in one which is irritative

Skew deviation of the eyes—in which for example one is directed upwards and the other downwards—occurs in certain lesions of the labyrinth 8th nerve and cerebellum

Examination of the Pupils

The following points must be noted about the pupils in every case —

1 **Size** —Compare the size of the two pupils first in a bright light and then in a dim light Note whether the pupils are large or small and whether any irregularity is present It must be remembered that the size of the pupil in health is subject to great variation As a rule the pupils are larger in dark eyes than in light They tend to be small in elderly subjects A much dilated pupil is often a sign of nervous exhaustion or instability Slight inequality of the pupils may also be present in perfectly healthy subjects

If one pupil is larger than the other one must decide which is the normal This is not always very easily answered but as a rule the pupil which exhibits the less mobility is to be regarded as the abnormal one

2 **Shape** —Note whether the pupil is circular in outline as it should be or whether its contour is irregular Such irregularities may be due to adhesion of the iris to the lens or to the effects of an old iritis Irregularity in shape of the pupil is often an early symptom in neurosyphilis

3 **Mobility** (*a*) **Reaction to light** —This is a reflex action. The afferent fibres involved are contained in the optic nerve travelling

The third or mandibular division is joined by the motor root. It supplies sensation to the lower part of the face, the lower lip, the ear, the tongue, and the lower teeth. It supplies also the salivary glands and through the motor division the muscles of mastication, the tensor tympani, and also perhaps the tensor palati, although many believe that this muscle is innervated by the spinal accessory.

2 Motor root.—This takes origin in a small nucleus lying internally to the chief sensory nucleus and partly also from the mesencephalic root which arises in nerve cells scattered around the aqueduct of Sylvius. It emerges at the side of the pons just in front of the sensory division, passes underneath the Gasserian ganglion and joins the inferior maxillary division, to which it gives its motor fibres.

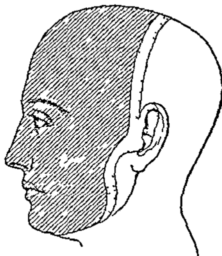


Fig 77.—Distribution of sensory loss in complete paralysis of the 5th nerve

(The sh d d a a rep e ent compl te anæsthesia th d tted a e part l d chiefly l ss to l ght t ch)

Paralysis of the whole 5th nerve leads to loss of sensation in the areas of skin and mucous membrane above mentioned, and to defective power of chewing (Fig 77). Trophic lesions may be present, and the salivary, buccal and lachrymal secretions much

admınished the sense of taste ıs occasionally abolished on the interior two thirds of the same side of the tongue

One curious result of the sensory paralysis is that the patient when drinking, imagines that the cup is broken, as he only feels it on one side of his mouth

How to test the fifth nerve 1 **Motor functions** —Ask the patient to clench his teeth while the observer keeps his hands on the temporal and then on the masseter muscles These should stand out with equal prominence on each side If there is paralysis on one side the muscles on that side will fail to become prominent On opening the mouth the jaw deviates towards the paralysed side, being pushed over by the healthy external pterygoid muscles The condition of the tensor tympani muscle cannot be satisfactorily examined except by noting whether there is any difficulty in hearing notes of a particular pitch—i e a diminution in the field of hearing

2 **Sensory functions** —The common sensibility of the area supplied is tested in the usual way (p 324)

Taste —In suspected lesions of the 5th nerve the sense of taste should always be examined as it seems probable that in certain cases at least taste fibres from the anterior two thirds of the tongue reach the brain through the 5th nerve As a rule, however, they pass from the lingual nerve into the chorda tympani, and thence through the geniculate ganglion and the nervus intermedius of Wrisberg into the medulla oblongata The taste fibres from the posterior third of the tongue enter by the glossopharyngeal nerve.

All the taste fibres enter the tractus solitarius and relay in the nucleus of the tract whence further fibres pass upward in the gustatory fillets to the thalamus and thence to the foot of the post central gyrus. *Ageusia* or loss of taste occurs with lesions of the peripheral pathways concerned or with centrally placed pontine lesions which may involve the gustatory fillets

How to test the sense of taste —Have some sugar some quinine and some salt all in powder Ask the patient to put out his tongue and to keep it out until the conclusion of the test Many men especially smokers are unable to taste on the protruded tongue The tongue should then be drawn in but the mouth kept open in order to avoid spread of the test substances Dry the tong

place some sugar on it rub it gently in and ask Is that salt? If taste is normal he will shake his head In this way all the substances are tried first on the anterior part of the tongue and then at the back. A weak galvanic current is also a useful test It should produce a sort of metallic taste

Alternatively use strong solutions of sugar and common salt and weak solutions of citric acid and quinine These are applied by a glass rod to the surface of the protruded tongue and if the taste is recognized the patient writes down sweet salt sour or bitter as the case may be without withdrawing the tongue After each test the mouth must be rinsed The quinine test should be applied last as its effect is more permanent than that of the others

Loss of taste may of course arise from lesions of the taste fibres in any part of their course above stated

In addition to loss of taste one should always ask the patient whether he has any abnormal taste sensations These may form the aura of an epileptic fit

3 **Reflex functions** —The corneal reflex (*see* p 331)

SEVENTH NERVE

Anatomy —The course of the fibres from the cortical centre to the nucleus of this nerve has already been described (p 251) The nucleus is situated in the pons externally to that of the 6th nerve. On leaving the nucleus the fibres wind round the nucleus of the 6th and finally emerge mesially to the 8th nerve between the olive and restiform bodies

The nerve lies in close contact with the 8th so that a lesion of the one at this part can hardly avoid injuring the other and enters the internal auditory meatus along with it During its course in the aqueduct of Fallopius it gives off a branch to the stapedius muscle and is joined by the chorda tympani, which contains taste fibres from the anterior two-thirds of the tongue In this part of its course the nerve is exposed to the effects of pressure owing to its being enclosed in a bony tube It emerges at a point opposite the junction of the anterior border of the mastoid with the ear and spreads out on the side of the face to supply its muscles.

Functions —The 7th is a purely motor nerve It supplies all the muscles of the face and scalp, except the levator palpebræ superioris. It also supplies the platysma

Effects of paralysis —These are usually at once seen on looking

at the patient The affected side of the face has lost its expression. The naso-labial fold is less pronounced the furrows of the brow are smoothed out, the eye is more widely open than the other and the mouth is somewhat drawn to the healthy side The patient is unable to whistle food is apt to collect between his teeth and his gums and saliva and any fluid he drinks may escape from the affected angle of the mouth

How to test the seventh nerve—1 Ask the patient to shut his eyes as tightly as he can Note that the affected eye is either not closed at all—in which case the eyeball rolls upwards to make up for the failure of the lid to descend—or if the eye is closed the eyelashes are not so far rolled in as on the healthy side Try also forcibly to open the eyes while the patient attempts to keep them closed If the orbicularis is acting normally it should be almost impossible to open the eye against the patient s wish If the muscle is partially paralysed however the exertion of very little force may open it.

The effect of screwing the eyes tightly shut causes the corners of the mouth to be drawn upwards In paralysis of the lower part of the face the corner on the affected side is either not drawn up at all, or not so much as on the healthy side

2 Ask the patient to whistle He is unable to do so

3 Ask him to smile or show his upper teeth The mouth is then drawn to the healthy side.

4 Ask him to inflate his mouth with air and blow out his cheeks Tap with the finger in turn on each inflated cheek Air can be made to escape from the mouth more easily on the weak or paralysed side

Signs of paralysis of the facial nerve in different parts of its course—Paralysis of the face presents different symptoms depending on whether the *lesion is situated above the nucleus at the nucleus or below it* The former constitutes what is known as upper motor neurone or supranuclear facial paralysis the latter produces lower motor neurone or infranuclear paralysis—

The chief difference between the two forms is that in *supranuclear paralysis* the lower part of the face is chiefly affected in *infranuclear paralysis* both the upper and lower parts are equally involved. The explanation of this is that there is bilateral

innervation of the muscles of the upper part of the face, and consequently a unilateral lesion only partially cuts off the nerve impulses to one side Sometimes a supranuclear lesion only affects the fibres concerned in emotional movement and this function should be tested separately from voluntary movement

Infranuclear facial paralysis may be produced by a lesion of the nucleus or of the facial nerve itself

A lesion inside the facial canal—unless it is towards the outer end—involves the fibres of the chorda tympani, and therefore produces loss of taste sensation in the anterior two thirds of the tongue

Lesions of the nucleus or the nerve below it will result in atrophy of the facial muscles Supranuclear lesions do not produce this effect Bilateral weakness of the face is sometimes difficult to detect especially if it is quite symmetrical

Abnormal facial movements—The muscles supplied by the facial nerve are frequently affected by spasm or spasmodic movements These may involve all the facial muscles or groups of them only The spasm may be of either the clonic or tonic variety (p 320) If present the nature of the movements their extent and the muscles affected by them should be carefully noted

EIGHTH NERVE (AUDITORY)

Anatomy—This nerve consists of two sets of fibres One set supplies the cochlea and subserves the function of hearing the other part supplies the vestibule and semicircular canals and is the nerve of equilibration The *auditory* fibres which arise from the cochlear ganglion enter the brain stem at the lower border of the pons and are distributed to the dorsal and ventral cochlear nuclei. The *vestibular* fibres take origin from the vestibular ganglion and terminate in a group of nuclei in the pons and medulla

The secondary auditory tracts after partial decussation, terminate in the inferior corpora quadrigemina and the medial geniculate bodies and another system that takes origin from these passes through the internal capsule to the cortical centre for hearing, in the 1st and 2nd temporo-sphenoidal convolutions Sounds received in one ear probably reach the opposite hemisphere of the brain predominantly but owing to the partial decussation of the secondary auditory tracts neither unilateral cerebral nor brain-stem lesions produce deafness in one ear

The vestibular nerve is closely connected with the cerebellum. It has cerebral connections but these are probably not very important.

Tests—1 Hearing—Before testing a patient s power of hearing exclude the presence of wax in the ear (*see* p 355) This done the hearing power can be tested by means of a watch Stand behind the patient and ask him to shut his eyes Begin outside the probable range of hearing power and bring the watch gradually nearer the ear asking the patient to speak when he hears the tick It is necessary to know at what distance the tick should be audible to a healthy ear Test each ear separately keeping one closed whilst the other is being examined

If impairment of hearing is detected it must be determined whether it is really due to disease of the auditory nerve or merely to some affection of the middle ear In order to settle this point the tuning fork test may be employed When the fork is beating strongly hold it opposite the ear if it can be heard then place its base on the mastoid process in order to determine if its vibrations can be heard when conducted through bone If the patient hears this ask him to compare the relative loudness of the fork when heard through air and through bone or to determine which can be heard the longer as the vibrations die out This is Rinne s test Normally, aerially conducted sounds are louder to the patient than those conducted through bone. In middle-ear disease, aerial conduction is diminished or lost while bone conduction remains more or less normal. When the auditory nerve is affected, both air and bone conduction are diminished or lost.

Weber s test though less reliable than Rinne s should also be used *Strike a tuning fork and place the end of it against the centre of the patient s forehead* If the deafness discovered by the watch is due to an affection of the middle ear the patient will hear the tuning fork *louder* on that side than on the healthy one. On the other hand if the deafness is due to disease of the auditory nerve the tuning fork will only be heard on the healthy side The test may also be carried out by means of the watch In affections of the nerve the watch is not heard even when pressed against the ear in disease of the middle ear it is heard even more loudly than when similarly applied to the healthy side The explanation of these facts is not yet clear nor are the tests invariably trustworthy They hold good however for most cases Other points in favour of the deafness being due to a lesion of the nerve and not to disease

of the middle ear are (a) if the hearing is better in a quiet place (b) if conversation is heard better than the watch (c) if inflation of the middle ear renders the hearing worse

Abnormal auditory sensations —The patient may complain of ringing in the ears or tinnitus The precise character of the sound varies in different cases It may be of a humming buzzing hammering or whistling character The presence or absence of this symptom should always be inquired for and whether it is constantly present or in what circumstances it occurs

Hyperæsthesia of the auditory nerve (hyperacusis) by which even slight sounds are heard with painful intensity sometimes occurs especially in hysteria and in lesions of the facial nerve above or in the aqueduct owing to paralysis of the stapedius muscle

Hallucinations of sound may also be present the patient fancying that he hears voices bells etc These occur chiefly in states of mental disturbance but they sometimes arise as the aura of an epileptic attack when the causal lesion is situated in or near the auditory cortex

2 **Vertigo** —The patient will describe this as giddiness or dizziness In order to constitute true vertigo external objects should seem to move round him Ask if this is the case and if so in what direction the objects seem to move Ask also whether the vertigo causes him to fall to the ground

Vertigo is usually due to an affection of the vestibular system, but weakness of an ocular muscle occasionally gives rise to it When a patient complains of vertigo one should look for signs of disease of the ear, 8th nerve, or brain stem.

NINTH (GLOSSO-PHARYNGEAL) TENTH (VAGUS) AND ELEVENTH (SPINAL ACCESSORY) NERVES

Anatomy —These arise in order from above downwards from an elongated nucleus in the floor of the 4th ventricle. They emerge by several roots along the lateral aspect of the medulla, beginning above in the groove between the olive and restiform bodies The spinal part of the 11th emerges from the lateral column of the cord, beginning as low as the 6th cervical nerve it passes up through the foramen magnum to join the medullary (accessory) part, and emerges with it through the jugular foramen After its emergence the two divisions of it again part company the medullary or accessory portion joining the vagus

The ninth (glosso pharyngeal) is sensory for the posterior third of the tongue and for the mucous membrane of the pharynx. It is motor for the middle constrictor of the pharynx and for the stylo-pharyngeus. It contains the taste fibres for the posterior part of the tongue (see p 303)

How to test the glosso pharyngeal —The 9th nerve is rarely paralysed alone Paralysis can best be diagnosed by examining its sensory and reflex functions Examine the power of taste in the posterior part of the tongue (p 303) Loss of it *may* mean paralysis of the trunk of the glosso pharyngeal nerve

Tickle the back of the pharynx, and note if reflex contraction occurs

The tenth (vagus) is motor for the soft palate (with the exception of the tensor palati) pharynx and larynx It is also sensory and motor for the respiratory passages the heart and (through the sympathetic ganglia) for most of the abdominal viscera

The fibres for the soft palate pharynx and larynx take origin in the nucleus ambiguus, emerge in the upper roots of the 11th reach the pharyngeal plexus and thence pass to the muscles of the palate the constrictors of the pharynx and to the larynx

The viscero motor and the cardio inhibitory fibres are derived from the dorsal vagus nucleus in the floor of the 4th ventricle

How to test the vagus —Paralysis of the vagus is chiefly made evident through its palatine and laryngeal branches

1 **The palate** —Ask the patient whether he is troubled with the regurgitation of fluids through his nose when he tries to swallow Thus is a common occurrence in total paralysis of the soft palate, owing to defective elevation during swallowing For a similar reason the patient is unable to pronounce words which require complete closure of the naso-pharynx Thus egg is sounded as eng, rub becomes rum, and so on In unilateral paralysis these symptoms are not observed

For direct examination of the soft palate place the patient facing the light with his mouth open and introduce a tongue depressor The position of the uvula is quite unreliable as a guide to the state of the soft palate as deviation of it is not uncommon even in health One must watch the movements of the palate during phonation

Ask the patient to say *Ah*, and observe whether both sides of the palate arch upwards In health elevation of the palate will occur when the patient says *Ah* If one side is paralysed that side will remain flat and immobile and the median raphe will be pulled towards the other side The manner in which the palate rises in such a case has been compared to the ascent of a curtain of which one string is broken In bilateral paralysis the whole palate remains motionless

2 The larynx —The superior laryngeal branch of the vagus is sensory for the larynx above the level of the true cords and is motor for the crico-thyroid muscle. Unilateral paralysis of the nerve does not produce any symptoms Bilateral paralysis causes the vocal cords to be relaxed The voice is therefore hoarse and deep and the utterance of high notes impossible

The recurrent laryngeal branch supplies sensation to the larynx below the level of the cords, and motor fibres to all the laryngeal muscles except the crico-thyroid Paralysis of it leads to appearances which are recognized by the laryngoscope and are described on p 361

The eleventh nerve —*Anatomy* —The accessory part of this nerve gives to the vagus motor fibres for the larynx and pharynx The spinal part of the nerve dips beneath the sterno-mastoid muscle about one inch below the tip of the mastoid process and emerges from underneath that muscle again at about the middle of its posterior border It supplies the sterno-mastoid and trapezius, which are also supplied by twigs from the cervical plexus. Lesions of the 11th nerve therefore lead to paralysis of these muscles

How to test the spinal accessory —Paralysis of the upper part of the trapezius is evinced by asking the patient to shrug his shoulders while the observer offers passive resistance by pressing on the shoulders from behind Paralysis of the sterno-mastoid causes weakness in rotation of the chin towards the opposite side

TWELFTH OR HYPOGLOSSAL NERVE

Anatomy —The 12th nerve arises from a nucleus in the lower part of the floor of the 4th ventricle close to the middle line It emerges between the anterior pyramid and the olive It is a purely motor nerve supplying the tongue and the depressors of the hyoid bone

Test —Ask the patient to put out his tongue as far as possible

If the hypoglossal is paralysed the tongue instead of being pro truded straight is pushed over to the paralysed side. Be careful not to mistake an apparent deviation of the tongue really due to the mouth being twisted to one side for a real deviation Such an apparent deviation occurs in facial paralysis Ask him also to move his tongue from side to side and to lick each cheek with it observe whether he can do so freely Note whether there is any wasting of the tongue and whether there is any tremor or fascicu lation in it The presence of wasting indicates that the lesion is either nuclear or infranuclear

Paralysis of the cervical sympathetic may be considered here A complete description of the functions and distribution of the nerve however is not necessary in such a work as this For purposes of diagnosis the fibres supplied to the eyeball are alone of import ance These take origin in the lower cervical and upper thoracic regions of the spinal cord (cilio spinal centre) from which the fibres emerge in the first thoracic nerve roots and pass to the sympathetic cord by the rami communicantes From the cervical sympathetic cord the fibres pass along the internal carotid to the cavernous plexus, and thence via the ophthalmic division of the 5th to the eyeball They convey the impulses which cause dilatation of the pupil, and supply also the unstriped muscle in the insertion of the levator palpebræ into the upper lid Paralysis of the cervical sympathetic is recognized by the following signs apparent enophthalmos, slight drooping of the upper lid, due to paralysis of the unstriped muscle fibres contained in it contraction of the pupil with absence of dilatation on shading the eye or on instillation of cocaine abolition of the cilio spinal reflex, less commonly absence of sweating even after the use of pilocarpine on the cor responding half of the head and neck both in front and behind extending as low as the 3rd rib and 3rd thoracic spine and over the whole of the upper limb on the same side Sweating of the face can best be induced by making the patient smell mustard

MOTOR FUNCTIONS

To examine the motor functions of a patient investigate five separate points —

1 The size of the muscles

2 The state of muscular tone
3 The muscular power
4 The co-ordination of movement
5 The presence or absence of involuntary movements

1 THE SIZE OF THE MUSCLES

The size of muscles is most easily ascertained when they are firmly contracted but may also be gauged by palpation Wasted or atrophic muscles are not only smaller but also softer and more flabby than normal When muscular wasting is accompanied by fibrosis the muscles feel hard and inelastic they become shortened and it is not possible passively to stretch them to a normal degree Contracture is then said to be present Contractures are also caused by prolonged hypertonus in a group of muscles Muscular atrophy is not only caused by neurological disorders Generalized muscular wasting is seen in patients with carcinoma and other chronic diseases Localized muscle atrophy may be due to injury or disease of a joint this is well seen in the thenar muscles in association with arthritis of the 1st metacarpo phalangeal joint or in the quadriceps in patients with affections of the knee joint In such instances muscular power is well preserved in relation to the degree of muscular wasting Some patients with muscular dystrophy develop large muscles (pseudo hypertrophy) due to pathological changes in the muscles themselves The calves, buttocks and infraspinati are particularly affected The enlarged muscles are weak in spite of their size

2 MUSCULAR TONE

Muscular tone is the state of tension or contraction that is always found in healthy muscles An increase in tone is spoken of as hypertonia and a diminution as hypotonia The degree of tone is estimated by handling the limbs and moving them passively at their various joints The maintenance of tone is dependent on a spinal arc afferent fibres entering the spinal cord and connecting with the anterior horn cells whence efferent fibres arise and pass to the muscles Tone is diminished or lost if this reflex arc is affected by disease Hypotonia therefore occurs in affections of the lower motor neurone or of the afferent sensory pathways (as in tabes)

Muscle tone is modified and regulated by impulses passing along

the pyramidal and extra pyramidal pathways The cerebellum is also closely concerned with the maintenance of tone.

Tone may be increased in states of anxiety It is reduced or lost in sleep and states of unconsciousness

Hypertonia follows lesions of the pyramidal system. This form of increased tone is termed spasticity and it has a characteristic distribution the upper limbs being fixed in flexion and the lower in extension. It must be remembered that plantar flexion of the foot is physiological extension. The resistance of the hypertonic muscles sometimes disappears suddenly on passive movement, the so-called clasp-knife spasticity When spasticity results from a partial lesion of the higher motor pathways in the spinal cord the lower limbs are paralysed in extension (paraplegia in extension) If the disease progresses and the lesion becomes complete the lower limbs adopt an attitude of flexion (paraplegia in flexion) this is the result of the uninhibited action of the spinal flexor reflex.

The hypertonia resulting from disease of the basal ganglia is termed rigidity. Here the muscular hypertonia is more uniform but often it is so distributed as to produce a general attitude of flexion of the limbs and trunk, e.g. paralysis agitans. The resistance to passive movement is fluctuant hence the use of the descriptive term cog wheel rigidity Occasionally a plastic type of rigidity is found with lesions of the mid brain or in catatonic states The resistance to passive movement is steady and continuous In hysterical rigidity the muscular resistance increases in proportion to the effort made by the observer to move the limb The muscles usually feel firmer than normal in hypertonia

When the muscles are hypotonic passive movement encounters little or no resistance and when the limb is handled or shaken the unsupported part flops about inertly Hypotonic muscles are abnormally soft to palpation The outstretched hypotonic upper limb usually shows an abnormal posture as in cerebellar disorders or chorea. It is hyperextended at the elbow, the forearm is over pronated, the wrist being unusually flexed, and the fingers over-extended at the metacarpo-phalangeal joints.

3 THE MUSCULAR POWER

Determine whether the patient is capable of performing gross muscular movements Can he walk? Can he :

Can he move each of his limbs as a whole?

Then investigate the range of the movements that the patient can make and the strength of the principal muscles and groups of muscles separately

The general rule in this investigation is to ask the patient to throw into action the particular muscle or group of muscles which one wishes to test whilst the observer offers to that action a greater or less degree of passive resistance The following is the method of procedure —

(i) Upper limb *Abductor pollicis brevis* —This is an important muscle clinically as it is the only intrinsic muscle of the hand invariably supplied by the median nerve which is easily tested The patient is asked to abduct his thumb in a plane at right angles to the palmar aspect of the index finger *The muscle can be seen and felt to contract*

Opponens pollicis —Ask the patient to touch the tip of his little finger with the point of his thumb

First dorsal interosseous —Ask the patient to abduct his index finger against resistance

Interossei and lumbricales —Test the patient s ability to flex his metacarpo phalangeal joints and to extend the distal phalanges The interossei also adduct and abduct the fingers When these muscles are paralysed and power is retained in the long flexors and extensors of the fingers the claw hand or main-en griffe deformity is produced The first phalanges are over extended and the distal two are flexed The fingers are slightly separated

Flexors of the fingers —Ask the patient to squeeze your fingers It is advisable to present him with two fingers only the middle finger being abducted to lie across the index or trauma to the examiner may result

Flexors of wrist —The hand being held with the palm upwards ask him to bring the points of his fingers towards the front of the forearm

Extensors of wrist —The hand being held with the palm down wards the observer grasps the patient s wrist and asks him to bend the hand up backwards as far as possible The fingers should be at the same time held flexed as the wrist can be extended by contraction of the long extensors of the fingers If he is unable to produce dorsiflexion of the wrist some weakness or paralysis of the extensors is present

Slight weakness of the extensors of the wrist may be elicited by asking the patient to grasp something firmly in his hand If the extensors are weak the wrist becomes flexed as he does so owing to the flexor muscles getting the better of the extensors

Weakness or paralysis of the extensors of the wrist leads to the condition known as **wrist-drop**

Brachio radialis —Place the arm midway between the prone and supine positions then ask the patient to bend up the forearm whilst the observer offers opposition to the act by grasping the hand If the muscle is healthy it will be seen and felt to stand out prominently at its upper part

Biceps —The patient s elbow being held against his side ask him to bend up the forearm while opposition is offered by grasping the hand or wrist If the biceps is healthy it will be observed to stand out prominently as it contracts

The *triceps* is tested by asking the patient to straighten out his forearm whilst the observer endeavours to keep it flexed by means of passive resistance

Supraspinatus —Ask the patient to lift his arm straight out at right angles to his side The first 30 of this movement are carried out by supraspinatus The remaining 60 is produced by the *Deltoid* whose anterior and posterior fibres help to draw the abducted arm forwards and backwards respectively

Infraspinatus —The patient is asked to tuck his elbow into his side with the forearm flexed to a right angle He is then instructed to rotate the limb outwards, the elbow being held against the side throughout The muscle can be seen and felt to contract

Pectorals —Ask the patient to stretch his arms out in front of him, and then to clap his hands while the observer endeavours to hold them apart Note whether both heads of the muscle are thrown into contraction or not

Serratus anterior —When this muscle is paralysed the scapula is winged, the vertebral border projecting The patient is unable to elevate his arm above a right angle, the deformity becoming more apparent as he tries to do so Pushing forwards with the hands against resistance also brings out the deformity

Latissimus dorsi —Ask the patient to clasp his hands behind his back while the observer standing behind the patient, offers passive resistance to the downward and backward movement or grasp

the two posterior axillary folds and ask the patient to cough In health the latissimus can be felt to contract

(ii) **Trunk muscles** —Weakness of the muscles of the abdomen is shown by the patient s inability to raise himself in bed without the aid of his arms *Babinski s rising up sign* consists in making the patient lie on his back with the legs extended and rise up without using his hands In organic spastic paralysis of a leg the affected limb will rise first, owing to the rigidity but in functional paralysis this does not occur. Paralysis of a portion of the anterior abdominal wall can be detected by the displacement of the umbilicus that occurs when the patient attempts to lift up his head from the pillow against resistance With paralysis of the lower segment the umbilicus moves upwards, but when the upper segment is affected the umbilicus is pulled downwards So also with unilateral paralysis the umbilicus is displaced by contraction of the unaffected muscle (*Beevor s sign*). To test the *erector spinae* and muscles of the back make the patient lie on his face and try to raise his head from the bed by extending the neck and back If the back muscles are healthy they will be seen to stand out prominently during this effort

The method of detecting paralysis of the *diaphragm* has already been described (p 188)

The *trapezius* is tested in its upper part by asking the patient to shrug his shoulders while the observer tries to press them down from behind In its lower part it can be tested by asking him to approximate the shoulder blades.

(iii) **The head muscles** —The methods of detecting weakness or paralysis in the muscles of the head have been referred to the section dealing with the investigation of the Cranial Nerves (p 278)

(iv) **The lower limb** —*The intrinsic muscles of the foot* are not usually examined in any detail When the interossei are weakened or paralysed a claw foot analogous to the claw hand may develop Rarely this deformity occurs in patients with spastic paraplegia of very long duration

Dorsi flexion and *plantar flexion* of the feet and toes are tested by asking the patient to elevate or depress the part against resistance

Eversion and *inversion* of the foot should also be investigated by

instructing the patient to turn the foot outwards or inwards against resistance

Extensors of knee —Bend up the patient s knee and then pressing with your hand on the sole of his foot ask him to try to straighten it out again

Flexors of knee —Raise his limb from the bed, supporting his thigh with your left hand and his ankle with your right Then ask him to try to bend his knee

Extensors of thigh —The knee being extended, lift the patient s foot off the bed and ask him *to depress it against resistance* If the extensors of the hip are paralysed he will be unable to do so

Flexors of thigh —The knee being extended, ask the patient to raise his leg off the bed.

The *adductors of the thigh* are tested by abducting the limb and then *asking the patient to bring it back to the middle line* while passive opposition is offered to the act In a similar way the *abductors* are tested by bringing the limb across the middle line and then asking the patient to move it outwards again

Rotators of thigh —With this lower limb extended on the bed ask the patient to roll it outwards or inwards whilst passive resistance is offered by grasping his foot

If on carrying out any of these tests a muscle or group of muscles is found to have only a feeble power of contraction **paresis** of it is said to be present If no contraction is elicited at all the condition is one of **paralysis** Apparent weakness may be due to simultaneous contraction of opposing muscle groups This can be detected by palpating the muscles at the same time as their power is tested This type of weakness is found in hysteria

The term **hemiplegia** is applied to a condition in which there is paralysis of one side of the face and of the arm and leg on the same side If the paralysis of the arm and leg is on one side, and that of some of the muscles supplied by the motor cranial nerves on the other, the condition is one of **crossed paralysis** The term **paraplegia** is applied to a paralysis of the lower part of the body; the term **monoplegia** to a paralysis of one arm (which is therefore characterized as a **brachial** monoplegia) one leg (**crural** mono plegia) or one side of the face (**facial** monoplegia)

The detection of hemiplegia in a patient who is comatose is often a very difficult matter However if the paralysis is of recent onset one can usually detect in such a patient a greater degree

of *limpness* in the paralysed limbs If his arm for example is raised from his side and allowed to drop it falls if it is paralysed as if it did not belong to him the sound arm also falls but not in such an utterly limp fashion The face is asymmetrical the angle of the mouth more open on the paralysed side and the affected cheek moves loosely outwards and inwards on respiration The abdominal and tendon reflexes may be abolished on both sides but an extensor plantar response is often obtained on the hemiplegic side

Myasthenic weakness that is weakness which becomes more marked after muscles have been exercised occurs in myasthenia gravis It is most commonly seen in the extra ocular muscles and those concerned with speech and swallowing

4 THE CO-ORDINATION OF MUSCULAR MOVEMENT

By muscular co ordination is meant the co operation of separate muscles or groups of muscles in order to accomplish a definite act If such co operation is absent or imperfect the performance of certain acts becomes difficult or impossible and the condition is then said to be one of **inco-ordination** The term **ataxia** or **ataxy** has a similar meaning

The co ordination of groups of muscles is the product of various factors among the chief of which are the afferent impulses coming from the muscles that never reach consciousness and those on which the sense of position of the limbs depends the state of tone of the muscles and in some acts perhaps cutaneous sensibility When inco ordination is present it is not always easy to say which of these factors is at fault The movements that constitute an act can be controlled and directed by vision but sight itself is not concerned in the co-ordination of movements When however there is loss of the sense of position the sensory defect may be compensated by vision and the disturbance of movement may become apparent only when the eyes are closed or bandaged or in the dark. Such ataxia occurs typically in tabes dorsalis, when sense of position is diminished or lost in the lower limbs

How to test co-ordination 1 *In the upper limbs* —Ask the patient to touch the point of his nose first with one forefinger and then with the other or ask him to bring the points of the two forefingers

together If he is able to succeed in these tests naturally and without making random shots no inco-ordination is present He may then be asked to perform the same actions with his eyes closed any additional irregularity of the movements can be due only to disturbance of the sense of position

Another good test in the upper limb is to ask the patient to thread a needle In this case the eyes must of course be left uncovered

2 *In the lower limbs* —If the patient is able to walk a good test in the lower limbs consists in asking him to walk along a straight line, e g the edge of a carpet If inco ordination is present he will soon deviate to one side or the other

If he cannot walk ask him as he lies in bed to place one heel on the opposite knee first with his eyes open and then when they are closed

Another method is to leave the eyes open and then ask him to follow with his toe one s forefinger as it describes circles in the air If he is able to describe the circles accurately his power of co ordination is good

Romberg s sign is often regarded as a special test for the co-ordination of the lower limbs, but though its presence is often evidence that the sense of position in these limbs is defective it may also be positive when the patient s instability is due to some other cause e g aural vertigo or a lesion of the cerebellum The patient is made to stand with his feet close together and if he can do so he then closes his eyes If the sign is present he begins at once to sway about or may even fall To elicit slight degrees of the phenomenon it may be necessary to make the patient stand on tiptoe with his knees bent. The essential feature of the sign is that the patient is more unsteady standing with his eyes closed than when they are open In sensory ataxia, caused for example by tabes dorsalis the patient is unable to maintain his attitude without the aid of vision on account of defective sense of position in the lower extremities

A special sign for cerebellar ataxia is adiadokokinesia it consists of inability to execute rapidly repeated movements In order to test for it the patient is asked to flex his elbows to a right angle and then supinate and pronate his forearms as rapidly as possible All normal persons can do this at approximately the same rate but as a rule slightly less rapidly with the left than with the right arm When however adiadokokinesia is present the movements are

slow, awkward and incomplete, and often become impossible after a few attempts

5 ABNORMAL MUSCULAR MOVEMENTS

These consist of involuntary muscular contractions of various sorts The first thing to note is whether the movements are widespread or localized

If they are confined to one part of the body note the joints at which the movements occur and the muscles or groups of muscles involved The term spasm is often applied to any exaggerated and involuntary muscular contraction The contraction may either be continuous in which case it is said to be tonic or there may be a series of short contractions with complete or partial relaxation of the mu cle in the intervals and in that case they are spoken of as clonic

Tetanic spasm is observed in its completest form in tetanus strychnine poisoning hydrophobia and some kinds of hysterical fits It may lead to a bending of the whole body backwards (*opisthotonos*) or sideways (*pleurothotonos*) or forward (*emprosthotonos*) The jaws may also be firmly clenched (*trismus*)

The term tetany is applied to a symptom-complex occurring under widely varying circumstances The underlying disturbance is a diminution in the ionized calcium in the serum There is a resultant hyper-excitability of the neuro muscular apparatus which manifests itself by intermitting spasms of the muscles

The e spasms may affect any muscles of the body but they most commonly occur in the periphery of the limbs They are usually bilateral in severe cases painful and they may last for ten to twenty minutes and recur over many weeks

An attack begins with a sensation of tingling and stiffness in the fingers The thumb is forcibly adducted the fingers pressed closely together being flexed at the metacarpo phalangeal joints and extended at the interphalangeal joints sometimes the index finger is more powerfully flexed than the other fingers the palm of the hand is made hollow by the approximation of its outer and inner margins the whole hand assuming a conical shape— accoucheur hand In severe cases the wrist and elbows may be flexed and the shoulders adducted When the lower limbs are affected the toes and ankles are plantar flexed the soles of the feet hollowed out and the knees and hips extended

Laryngospasm may occur in tetany In rickets it is especially common and often appears without the occurrence of tetany It is commonly termed *laryngismus stridulus* At any time in the night or day the child affected will hold its breath until the face is cyanosed Then the momentary spasm of the glottis relaxes and as it does so air is drawn past the still closely approximated vocal cords with a high pitched crowing sound

In tetany the muscles of the trunk are involved only rarely those of the face are sometimes affected the lips being pursed up — carp mouth —and spasm of the oculo motor muscles sufficient to cause diplopia is occasionally seen

The following physical signs peculiar to tetany are important the more so since they often persist between attacks —

Trousseau s sign —Pressure upon the vessels or nerves of the limb for example by a tourniquet or sphygmomanometer bag will produce the typical spasm (main d accoucheur) or will augment it if it is already present

Chvostek s sign —Tapping over muscles and over nerves super ficially situated induces spasm For example the light tap of a *patella hammer over the facial nerve in front of the lobe of the* ear causes muscular twitchings over the whole of that side of the face

Erb s sign —Owing to increased electrical excitability of the motor nerves fibrillation and spasm may be induced in the muscles by cathodal stimulation with currents subminimal to the normal subject

Occupational cramps *may occur in persons whose occupations* involve complicated movements of the fingers for long periods of time for example telegraphists clerks and violinists They are psychogenic

Conjugate ocular spasms or oculo gyric crises sometimes occur as a sequel of encephalitis lethargica They consist of attacks of spasmodic conjugate ocular deviation usually upwards which last about half an hour During the attack the patient is unable to deviate the eyes downwards below the horizontal plane and any attempt at downward displacement is associated with intense tremor of the lids

Clonic spasms are of various degrees of severity If very wide spread they are spoken of as **convulsions** and are seen typically in epilepsy If the patient gives a history of fits their character

should be inquired into following the lines laid down on p 8 Should the observer be fortunate enough to witness an attack he should note—

i *The nature and distribution of the movements* —Are they general or confined to one limb or part of a limb? What part is first and what last affected? Are the convulsions tonic or clonic? Is there any struggling arching of the back or attitudinizing? Are the abdominal muscles involved or not?

ii Is there any *involuntary evacuation* of the bladder or rectum? Is there any blood or froth about the mouth? Does the patient change in colour?

iii *The state of the eyes*—Are the eyes open? Is the corneal reflex present or abolished? Do the pupils react to light? Is there any inco-ordinate movement of the eyeballs?

iv How does the patient behave *after the fit*?

If one group of muscles is first affected the spasm spreading to others by degrees it indicates a spread of the discharge along the cortex cerebri This occurs typically in Jacksonian epilepsy

Myoclonus is the term given to shock like contractions occurring in individual muscles They have been observed in certain forms of encephalitis and are also encountered as manifestations of minor epilepsy Their frequency varies from 10 to 80 contractions a minute but they usually have no definite rhythm

Tremor consists of more or less rhythmical oscillations of a part or parts of a limb and is due to the alternate contractions of a group of muscles and its antagonists Tremor may be either *fine* or *coarse* Fine tremor is usually more easily felt than seen It occurs in exophthalmic goitre alcoholism and in some forms of metallic poisoning All forms of tremor are most easily seen by increasing the leverage at which the affected muscles act Thus tremor of the upper limbs is often brought out by getting the patient to extend his arms in front of him In describing tremor always note whether it is constantly present or if it is affected in any way by voluntary muscular action Also observe its rate the amplitude of the movements and whether they are regular or irregular Ask the patient to lift a glass of water to his lips, and note whether the tremor is increased thereby (as it is in cases of cerebellar ataxia), or whether it is diminished or altogether abolished

Tremor which only comes on when the patient attempts to use the affected muscles is described as *intention tremor*

Clonic contraction of bundles of fibres in a muscle is termed **fasciculation** It is seen in many cases of progressive muscular atrophy, and indicates an abnormal state of nutrition in the anterior horn cells connected with the affected fibres The term **fibrillation** is reserved for contractions of individual muscle fibres *Such movements cannot be detected clinically* but may be recorded by the electromyograph

To the transient flickering of a few muscle fibres (commonly known as live flesh or live blood) the term **myokymia** is applied It is most often seen in the orbiculares palpebrarum and is usually an indication of fatigue or debility It also occurs as an independent condition and is then more or less general

The term choreic is applied to involuntary movements of a quasi-purposive character occurring in individual muscles or groups of muscles Such movements are seen most typically in chorea *minor or St Vitus s dance They consist of abrupt involuntary* twitchings or contractions which cause the patient (usually a child) to seem fidgety and unsettled They are increased by mental agitation but are often diminished by voluntary muscular effort

If the movements are limited to one side of the body the term *hemichorea* is applied

Choreic movements if slight can be elicited in two ways First one may ask the patient to hold both hands straight up above the head or second one may ask him to spread out his hands palms downwards on the extended hands of the observer In the former case it may be observed that the patient is unable to hold up his hands steadily for any length of time in the latter one may notice that little twitchy movements soon become evident in the patient s fingers.

If the patient is unable to write one may get him to scrawl his name with the affected hand and keep the result for purposes of comparison later In this way one is able to estimate any increase or diminution in the choreic movements

Choreiform movements also occur as a result of cerebral disease they are then usually limited to one side of the body and as they appear after local lesions they are sometimes described as *post hemiplegic chorea* Hemichorea without hemiplegia is seen in lesions of the corpus luysii

Tics are co ordinated, repetitive, purposive acts which are started in the first place by some external cause or by an idea

repetition they become habitual and finally involuntary without any relation to the cause that first excited them They may assume various forms perhaps the most common are blinking of the eyes smacking the lips or rotation or nodding of the head They can be distinguished from other involuntary movements by their complexity and by their always retaining their purposive character

The term athetosis is used to describe slow muscular contractions which lead to continuous and deliberate twisting movements specially affecting the hands and feet but sometimes extending to involve the whole limbs and even the trunk

The last point to be noted regarding any abnormal muscular movement is whether or not it persists during sleep

SENSORY FUNCTIONS

In investigating the sensory functions of a patient we have to test the acuteness of the following forms of sensibility —

1 Tactile sensibility This includes the powers of appreciating light touch and pressure and of tactile localization and *discrimination*
2 Sensibility to pain
3 Thermal sensibility
4 The sense of position and the appreciation of passive movement
5 The recognition of the size shape weight and form of objects
6 The appreciation of vibration

In addition the presence or absence of any abnormal sensations is noted

At the outset explain to the patient the nature of the tests to be performed and so secure as far as possible his intelligent co operation The eyes should then be closed or the part under examination screened from sight and the different forms of sensibility tested as follows —

1 Tactile sensibility —Use a wisp of cotton wool or a fine camel hair brush If it is desired to test the sensibility of the skin to light touch over a hairy part it is essential to shave it as the sensibility of the hairs themselves is so acute

Tell the patient to say Now every time he feels a touch

Compare corresponding points on opposite sides of the body and employ every now and then a negative test asking the patient if he feels you touch him in order to prevent his making random replies The appreciation of pressure touch should then be tested this may be done by touching him with the point of a finger or any blunt object. It is important that its temperature should not differ much from that of the skin and the pressure must not be *so heavy as to give pain or discomfort. Ask him also to localize* the stimulus by describing or in other way indicating the exact position of the spot touched This is important as a patient may be able to feel the stimulus and yet not be able to localize it.

Sensibility to touch may be altered in various ways (1) It may be entirely abolished. This constitutes anæsthesia If the abolition affects the whole of one side of the body it is termed *hemianæsthesia* If the existence of anæsthesia is discovered one must at once proceed to mark out its exact extent and boundaries Partial loss of tactile sensation is called hypoæsthesia (2) Sensibility may be so altered that what should in health be felt as a mere touch produces a painful impression resembling pricking or burning This is generally called hyperæsthesia Hyperæsthetic spots are sometimes met with especially in hysterical patients The commonest sites for these are over the brim of the pelvis in the inframammary region, along the vertebral column and on the scalp Pressure on such spots may sometimes induce hysterical fits (3) Sensation may be appreciated well enough but there may be great delay in its conduction, an appreciable interval occurring between the application of the stimulus and the response of the patient. This delayed conduction exists not infrequently in cases of tabes (4) The stimulus may be badly localized the patient believing for example that the outer side of a limb was touched when the stimulus was really applied to its inner aspect. Sometimes a touch on one side of the body is referred to a corresponding point on the opposite side this is termed allocheiria

Tactile discrimination Ability to discriminate between two points is tested with Weber's compasses The patient is asked whether he is being touched with one or both points of the compasses Normally 1 cm of separation of the points is appreciated on the palmar surfaces of the thumb and fingers

2 Sensibility to pain —Pain may be evoked either by a cutaneous

stimulus as the prick of a pin or by pressure on the deeper structures as the muscles or bones Sensibility to superficial and to pressure pain should be tested separately

(a) Superficial pain —The point of a steel pin or needle may be used as the stimulus Care must be taken that the patient distinguishes between the *sharpness of the point* (that is its relative size) and the *pain* which the prick evokes it often happens that even when sensibility to pain is abolished he can recognize that the stimulus is pointed and thus confuse the observer by calling it sharp

(b) Pressure pain is examined by squeezing the muscles or the tendo Achilles Abolition of pressure pain is often the most prominent sensory disturbance in tabes dorsalis

Absence of sensibility to pain is termed analgesia partial loss of pain sensibility is called hypoalgesia and an exaggerated sensibility so that even a mild stimulus causes an unnatural degree of suffering is known as hyperalgesia

3 Thermal sensibility is conveniently examined by using test tubes containing hot and cold water The part to be tested is touched with each in turn and the patient says whether each tube feels hot or cold It is often important to determine the thresholds for heat and cold i e the lowest temperature that feels warm and the highest that is cold This can be done by noting the temperatures of the water in the tubes on thermometers contained in them to do this it is better to use large copper or glass test tubes Note also the reactions evoked by high and low temperatures and the sensations they produce in the patient It frequently happens that such temperatures evoke only pain and may be called indiscriminately hot or cold

The different forms of sensibility already mentioned may have to be tested on mucous membranes as well as on the skin surfaces The sensibility of some viscera is also important Thus the absence of pain on squeezing the testicle may be an early sign of tabes

4 Sense of position —The patient s eyes being carefully shut take hold of one of his limbs and move it about in various directions through the air finally leaving it in some definite position say semi flexed and slightly elevated then ask him to put the corresponding

limb in a similar position If there is no paralysis of the latter and yet the patient is unable to imitate with it the position of the other then there is reason to believe that the sense of position is impaired

In the case of the hand the patient may be told that the fingers of one hand will be moved and that he must imitate with the other the position in which they have been placed In the case of the foot he may be told that the great toe will be placed pointing upwards or downwards and that he must try to tell which it is

In testing a patient s sense of position in this manner be careful not to allow the part tested to touch any other skin surface otherwise the patient will be able to appreciate its position by the information derived from his ordinary sense of touch

A very delicate test for the sense of position in the upper limbs consists in shutting the patient s eyes and then making him hold his arms straight out in front of him with the fingers in a horizontal row After a moment or two if the muscular sense is defective the fingers cease to remain in an even line Some will rise a little others fall or even become twisted in below the rest

The appreciation of movement is closely related to the sense of position and should be tested at the same time Grasp any segment of a limb firmly and then move it gradually into another position ask the patient to say Now as soon as he recognizes the movement and note the angle through which the limb was moved If the appreciation of movement is diminished this angle is many times greater than that which is necessary in a normal limb but if the defect is slight it may be necessary to measure the range of the movement accurately for comparison Movements of 10 can be appreciated at all normal joints Finally test if the patient can recognize the direction of the movement that is whether the joint is flexed or extended. It often happens that the patient can recognize the occurrence of a movement though he is ignorant of its direction

5 **The recognition of size shape and form** —These faculties can be tested most accurately in the hands To test size place in the patient s palm objects of the same shape but of different sizes as small rods or matches of different length Two objects should be applied consecutively and he is asked to say which is the larger

To test the power of recognizing form familiar objects as coins

a pencil a penknife scissors etc are placed in the hand and the patient is asked to identify them or to describe their form Loss of this power is generally known as astereognosis

6 **Appreciation of vibration** —If the foot of a vibrating tuning fork is placed on the surface of the body the vibrations can be felt provided they are sufficiently strong This is a valuable test as the ability to appreciate vibration may be lost in various diseases as in tabes dorsalis peripheral neuritis and conditions that involve the posterior columns of the cord. A heavy tuning fork of 128 vibrations a second (C o) is generally employed Make the fork vibrate by striking its prongs gently on a firm object and place its foot immediately on the part to be tested ascertain if the patient perceives the vibrations and if so ask him to say at once when he ceases to feel them If the fork is then transferred to the observer it can be seen if the sensibility to vibration is diminished or not and the amount of diminution can be measured by the further time that the vibration can be perceived by the normal parts The appreciation of vibration is sometimes spoken of as **pallæsthesia**

Are there any abnormal sensations present?—These are termed **paræsthesiæ** and consist in various sensations experienced by the patient in the absence of any outward stimulus The commonest of these are a feeling of pins and needles of numbness of heats or chills of pressure or tightness of itching—sometimes termed pruritus—or a feeling as if insects were crawling over the body (formication)

Sensory inattention —This phenomenon is sometimes found in patients with lesions of the parietal lobes, it is demonstrated as follows The patient is asked to close his eyes Mirror points on opposite sides of the body are then tested simultaneously with identical stimuli, either painful tactile or thermal The patient is asked to say which side is being stimulated If there is sensory inattention he will fail to appreciate the stimulus on the affected side of the body that is the side opposite to his lesion Sensory inattention may be found in the absence of any formal sensory loss and is thus a valuable sign Visual inattention is tested along similar lines the examiner using simultaneous movements of his

index fingers in opposite fields as competing stimuli The patient with visual inattention fails to notice the movement of the finger in the affected field This again may occur in the absence of any formal field defect

REFLEXES

There are three types of reflex —

1 The superficial reflexes
2 The deep or tendon reflexes
3 The organic reflexes (including the action of the sphincters)

I SUPERFICIAL REFLEXES

Here the simplest form of reflex action is concerned On stimulation of a certain part of skin or mucous membrane contraction of certain muscles results The path of the impulse is by the sensory nerve fibres to the grey matter of the cord or to a higher centre in the brain stem or forebrain thence by motor nerve fibres to the muscle A lesion in any part of this path causes the reflex to disappear Thus anæsthesia of the skin disease of the sensory fibres or posterior nerve roots changes in the grey matter of the cord lesions of the motor nerve fibres or of the fibres of the muscles may all cause abolition of the superficial reflexes In addition the reflex excitability of some individuals is normally much greater than that of others which makes it difficult for estimation of the value of slight alteration in the reflexes to be made unless the lesion is unilateral in which case the healthy side can be taken as a standard of comparison The investigation of the superficial reflexes is of value as affording information regarding the health of the reflex arc concerned and as a guide to the presence or absence of disease elsewhere In hemiplegia the superficial reflexes are disturbed on the paralysed side

The chief superficial reflexes of spinal origin their nature the mode of obtaining them and the level of the cord concerned in their production, are given in the table on p 332

The plantar reflex demands special consideration To elicit it the muscles of the lower limb should be relaxed and care should be taken that the sole of the foot is warm The outer edge of sole of the foot is stimulated by gentle scratching with a Yale

or a pin In healthy adults a minimal stimulus produces a contraction of the tensor fascia lata often accompanied by a slighter contraction of the adductors of the thigh and sartorius With a slightly stronger stimulus flexion of the four outer toes appears which increases with the strength of the stimulus till all the toes are flexed on the metatarsus and drawn together the ankle being dorsiflexed and inverted With still stronger stimuli violent regular movements of the limb occur which spread to the lower part of the trunk and to the opposite side

It is doubtful whether the plantar reflex is ever completely absent in healthy subjects

In infants between six and twelve months old the reflex is very brisk and differs markedly from that in adults The earliest response is in the great toe which is drawn back This is followed by extension and spreading out of all the toes with eversion of the foot or dorsiflexion of the ankle and subsequently by flexion of the hip and knee (infantile response)

During sleep the plantar reflexes are diminished and the infantile and adult form preserved save in some children up to the age of 12 years to whom in deep sleep the infantile form of reflex returns In pathological conditions the reflex varies and may be of great diagnostic importance In *lesions of the pyramidal systems* an alteration in the response was first described by Babinski In this which is spoken of as Babinski s sign or the extensor response the reflex closely resembles that obtained in infants but differs in a few points The whole response is more deliberate than that obtained normally either in adults or infants and appears with much more certainty than does the flexor response to each stimulation Extension of the great toe precedes all other movement It is followed by spreading out and extension of the other toes dorsiflexion of the ankle and flexion of hip and knee The small amount of movement at the ankle is less conspicuous than the brisk movement in the normal response The extensor response is most easily elicited by stimulation of the outer part of the sole and with slight pyramidal lesions may be evoked from this region alone when a normal flexor response is obtained by stimulating the inner part If the lesion is progressive the area in which the extensor plantar reflex can be excited (receptive field) increases and spreads first inwards over the sole of the foot and then upwards along the leg to the knee or even the groin For this reason

extension of the great toe generally associated with some dorsi flexion of the foot can often be obtained by squeezing the calf or pressing heavily along the inner border of the tibia (**Oppenheim s sign**) or by pinching the tendo Achilles (**Gordon s reflex**) when the upper motor neurones are injured or diseased The extensor response is met with in adults only in cases of organic disease involving the pyramidal tract

The **superficial abdominal reflexes** (*see* p 332) are also valuable signs as they disappear when the pyramidal tract of the same side is in any way affected The ower abdominal reflex is then abolished earlier than the upper It is often impossible to obtain them in old or obese people or in women who have borne many children

The following superficial reflexes are dependent on cranial nerves —

i **Conjunctival** —Elicited by touching the conjunctiva resulting in contraction of the orbiculares palepbrarum The nerves concerned are the 5th (sensory) and the 7th (motor)

ii **Pupil reflexes** —(*See* p 299)

iii **Corneal reflex** —Consists in rapid closure of the eyelids on touching the cornea (e g with cotton wool) It depends upon the integrity of the first division of the 5th cranial nerve on the afferent side and upon that of both 7th cranial nerves on the efferent

iv **Palate reflex** —Elevation of the palate on touching the mucous membrane covering it The nerves concerned are the trigeminal and glosso pharyngeal on the afferent side and the vagus on the efferent

2 DEEP OR TENDON REFLEXES

If a muscle is put upon the stretch and its tendon is sharply struck the muscle immediately contracts This is spoken of as a *deep* or tendon reflex The tendon reflexes are dependent upon the integrity of spinal reflex arcs consisting of afferent and efferent pathways The upper motor neurones exert an inhibitory effect on these reflex arcs and when this influence is cut off the tendon reflexes are increased

Exaggeration of the tendon reflexes unless some other disease

CHIEF SUPERFICIAL REFLEXES OF SPINAL ORIGIN

Refl x	How exc t d	Res lt	Le el of cord concern d
Anal	Stroking or scratching the skin near the anus	Contraction of the anal sphincter	3rd and 4th sacral segments
Bulbo-cavernosus	Pinching dorsum of glans penis	Contraction of bulbo cavernosus	3rd and 4th sacral segments
Plantar	Stroking sole of foot	Movements of toes of toes and foot or leg	Lower part of lumbar enlarge ment (5th lumbar and 1st sacral segments)
Cremasteric	Stroking skin at upper and inner part of thigh	Drawing upwards of testicle	1st and 2nd lumbar segments
Abdominal	Stroking abdominal wall below costal margin at level of umbilicus and in iliac fossa	Contraction of abdominal muscles	7th to 12th thoracic segments
Scapular	Stroking skin in inter scapular region	Contraction of scapular muscles	5th cervical to 1st thoracic segment

The cremasteric reflex can often be most easily elicited by pressing over the sartorius in the lower third of Hunter s canal

coexists is always associated with lesions of the upper motor neurones—i e with lesions affecting either the motor cortex or the fibres passing from it to the anterior horns of the cord A similar exaggeration may be brought about by anything that stimulates the reflex arc as strychnine the toxin of tetanus exposure to cold and other factors Attention or expectation can also induce a state of greater excitability and the reflex response is greater if the muscles concerned are not fully relaxed this is probably the explanation of the frequent exaggeration of these reflexes in hysteria and other functional conditions Increase of the tendon reflexes is consequently not invariably a sign of organic disease

On the other hand anything that impairs the activity of the reflex arc makes it correspondingly difficult to elicit the tendon reflexes Their diminution or abolition is therefore always associated with disease of the lower afferent and efferent neurones or of the reflex centres in the grey matter of the spinal cord Hence it is that in tabes dorsalis in which the posterior roots are involved and in peripheral neuritis in which both motor and sensory fibres are generally affected the deep reflexes are absent

The tendon reflexes may be normal in cerebellar disease but sometimes they are altered particularly the knee jerk which is pendular in quality

In a lesion—e g a fracture-dislocation—which produced complete transverse destruction of the cord at any level one might expect that owing to the cerebral influences being cut off all the deep reflexes below that level would be exaggerated but for the first few weeks at least all the reflexes are totally abolished This seems to be due to a state of spinal shock in which the activities of the isolated portion of the cord are depressed as a result of its severance from the rest of the central nervous system Later however if the general condition of the patient is satisfactory the reflexes reappear and a condition may develop in which a single stimulus is capable of producing a widespread effect Thus if the spinal cord is divided in the midthoracic region stimulation of the abdomen or lower limbs may evoke a bilateral flexor spasm of these parts along with reflex evacuation of urine and perhaps fæces and an outburst of sweat from the lower limbs and trunk This reaction has been called a mass reflex But if bedsores cystitis or serious nutritional disturbances develop pr general debility the reflexes may remain absent or disappear

The Knee-jerk or patellar tendon reflex is the best known of the deep reflexes It consists in a contraction of the quadriceps extensor when the patellar tendon is tapped The spinal segments concerned are the 2nd 3rd and 4th lumbar It is best tested with the patient supine The examiner s hand is passed under the knee to be tested and brought to rest upon the opposite knee the knee to be tested rests on the dorsum of the observer s wrist Now distract the patient s attention in some way and strike the quadriceps tendon midway between its origin and insertion with a patellar hammer Following the blow there will be extension of the knee from contraction of the quadriceps

The briskness of the knee jerk varies greatly in different individuals In health it is hardly ever entirely absent Sometimes one is unable to elicit it without applying **reinforcement** This is done by asking the patient to make some strong voluntary muscular effort with the upper limbs for example to hook the fingers of the two hands together and then to pull them against one another as hard as possible While he is doing this a further attempt is made to elicit the knee jerk which is often successful Reinforcement acts by increasing the muscular tone throughout the body

The following tendon reflexes are similar in nature to the knee jerk but they are connected with the spinal cord through anterior and posterior roots at different levels and so are valuable in diagnosis

Ankle-jerk —Place the lower limb on the bed so that it lies everted and slightly flexed Then with one hand slightly dorsiflex the foot so as to put the tendo Achilles on the stretch and with the other hand strike the latter on its posterior surface A sharp contraction of the calf muscles results This reflex can also be conveniently elicited when the patient is kneeling on a chair It depends upon the 1st and 2nd sacral segments

Triceps jerk —Flex the elbow then tap the triceps tendon just above the olecranon The triceps contracts The reflex depends upon the 6th and 7th cervical segments

Biceps jerk —The elbow is flexed to a right angle and the forearm placed in a semipronated position the examiner then places his thumb on the biceps tendon and strikes it with the patellar hammer

The biceps contracts The 5th and 6th cervical segments of the cord are concerned

Radial supinator jerk —A blow upon the styloid process of the radius produces flexion of the elbow This reflex also depends on the 5th and 6th cervical segments With lesions at this level the reflex is abolished but when it is tested flexion of the fingers may result from stretching of the flexor muscles in the forearm by the blow This phenomenon is known as inversion of the radial (supinator) reflex

Jaw jerk —Ask the patient to open his mouth but not too widely Place one finger firmly on his chin and then tap it suddenly with the other hand as in percussion A contraction of the muscles that close the jaw results This jerk is sometimes absent in health and is increased in upper motor neurone lesions above the 5th nerve nuclei

The phenomenon of **clonus** is often elicitable when the tendon reflexes are exaggerated as a result of a pyramidal lesion

Ankle clonus —Bend the patient s knee slightly and support it with one hand grasp the fore part of the foot with the other hand and suddenly dorsiflex the foot The sudden strain put upon the calf muscles causes them to contract The pressure of the hand upon the sole of the foot is meanwhile continued and when the contraction ceases causes the muscle again to become tense and so produces another contraction in the latter In this way a whole series of contractions—i e a clonus—results

The relative tendency to the development of ankle-clonus on the two sides is best estimated by slowly dorsiflexing the foot and observing the exact point at which the movements first begin The less the degree of dorsiflexion required to produce the clonus the greater is the tendency to the development of the latter

In cases of functional paralysis a spurious clonus may be elicited It is usually ill sustained and irregular in rhythm and can be recognized by the feeling of voluntary contraction in the muscles especially at the beginning of the clonus

Sustained ankle-clonus is nearly always a sign of pyramidal disease The spinal segments concerned in it are the 1st to 3rd sacral

Patella clonus —In cases where the knee jerk is exaggerated one can sometimes elicit a patella-clonus by extending the patient's leg and then suddenly pushing down the patella towards the foot If the pressure on the latter is continued a series of clonic contractions of the quadriceps can in many cases be produced

3 ORGANIC REFLEXES AND SPHINCTERS

This term includes the reflexes governing such processes as respiration deglutition micturition and defæcation They depend upon complex muscular movements excited by increased tension in the wall of the viscus concerned or in the case of respiration partly by stimulation of a centre in the medulla

One should ascertain from the patient whether he has any difficulty in swallowing noting especially whether there is any regurgitation of food through the nose The function of deglutition does not usually require to be specially tested beyond the examination necessary to exclude the existence of an obstruction

Defæcation —The patient should be questioned as to any difficulty in the act and as to the presence of rectal sensation Note also the occurrence or not of incontinence of fæces

The reflex action of the *anal sphincter* may be tested by introducing the oiled gloved finger into the anus and noting whether contraction of the sphincter occurs with normal force whether it is weak or altogether inactive or whether any spasm is excited

The activity of the anal sphincter reflex may also be tested by pricking the skin in the neighbourhood of the anus If the conditions are normal a brisk contraction of the sphincter should immediately be visible This depends upon the 4th and 5th sacral segments

Micturition —The patient should be questioned as to difficulty or pain in the act (*see* p 8) He should be asked whether bladder and urethral sensation are normal Then note whether there is either *retention* or *incontinence* of *urine* If there is incontinence ascertain by the use of the catheter whether it is due to the *overflow* from a distended bladder or whether it is a *reflex incontinence*—i e whether the bladder merely fills up and then empties itself completely by reflex action In another group of cases the patient feels the desire to micturate and is unable to restrain the act

which takes place at once This is spoken of as *precipitate micturition*

The bladder (Fig 78) receives a double innervation from the

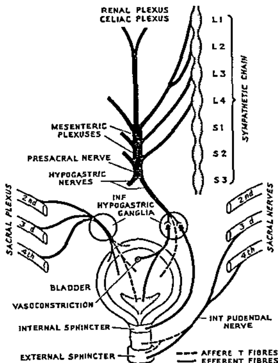


Fig 78 —Diagram to show afferent and efferent pathways to the bladder and sphincters Post ganglionic parasympathetic neurones are omitted (*Aft M Lell*)

spinal cord (1) sympathetic fibres from the 11th and 12th thoracic and 1st lumbar segments by way of the hypogastric nerves and (2) parasympathetic fibres from the 2nd 3rd and 4th sacral segments by way of the pelvic nerves The parasympathetic fibres thought to be of major importance Sensory impulses from

bladder and urethra are carried to the brain via the spino thalamic tracts The pathways concerned with the voluntary initiation of micturition probably run downwards from the motor cortex in the posterior marginal part of the lateral columns of the spinal cord The rectum along with the whole of the large intestine is also innervated by the pelvic nerves from the 2nd 3rd and 4th sacral segments

TROPHIC DISORDERS

In disease of the nervous system the nutrition of different tissues or organs may be impaired The *bones* may become more brittle or may exhibit spontaneous fracture (osteopathies) or the *joints* may be the seat of painless effusion with or without atrophy or enlargement of the articular ends of the bones (arthropathies) In other cases the bones and joints are involved together (osteo-arthropathies) More commonly the *skin* is the seat of change It may exhibit an erythema which may pass on to ulceration and the formation of bedsores at points of pressure pigmentary changes may develop or various eruptions—urticarial vesicular pemphigoid or herpetic—or the skin may be simply glossy Perforating ulcers may appear usually on the toes or soles of the feet as in tabes or there may be actual gangrene or the development of painless whitlows In other cases it is the epidermic appendages which especially suffer change the hair falling out or the nails becoming dry and brittle Atrophy of the *muscles* is a common phenomenon

SIGNS OF MENINGEAL IRRITATION

1 Neck rigidity —*To test* for neck rigidity the examiner places his hand behind the patient s occiput and flexes the head so that the chin touches the chest Normally this movement can be carried out without pain In meningeal irritation the test causes pain in the neck sometimes radiating down the back and the movement is resisted by spasm in the extensor muscles of the neck Neck rigidity is also caused by diseases of the cervical spine Head retraction is an extensive degree of neck rigidity

2 Kernig s sign is tested by attempting to carry out passive extension of the patient s knee when his hip is fully flexed The test

causes pain and spasm of the hamstrings in meningeal irritation affecting the lower part of the spinal sub arachnoid space If Kernig's sign is positive in the absence of neck rigidity the inference may be drawn that the patient is suffering from spinal meningeal irritation

These two tests depend upon the fact that stretching the spinal nerve roots in conditions of lepto meningeal irritation causes a reflex muscular spasm They are positive in meningitis sub-arachnoid hæmorrhage but also in patients with meningism a state of irritation of the meninges seen most commonly in young children with acute specific fevers

3 **Straight leg raising** —This test is used in patients with sciatica The sciatic nerve and its component roots are stretched by passively elevating the patient s extended leg with the examiner s hand which is placed behind the heel The movement is restricted by pain in conditions in which the nerve or as more frequently happens its spinal roots are involved

ANCILLARY INVESTIGATIONS

The following special methods of investigation are in common use in neurological practice

1 **Electro-encephalography** —The electro-encephalogram (E E G) is a record of the electrical activity of the cerebral cortex Elec trodes applied to the patient s scalp pick up the small changes of electrical potential which are constantly taking place in the cortex After amplification these changes are recorded on a strip of paper Electro-encephalography is of particular value in the investigation of epileptic patients and in the localization of cerebral tumours *and other expanding intracranial lesions*

2 **Electro myography** —This consists in recording the amplified electrical activity of muscle The investigation is of assistance in the diagnosis of lesions of the lower motor neurone and of diseases of muscle

3 **Neuro-radiological diagnostic procedures** —Apart from the use of plain X rays the following methods are often employed

(a) *Air studies*—The ventricular system is outlined with air and radiograms of the skull are then taken The size shape and position of the ventricles may be altered by expanding intracranial lesions Atrophic changes in the brain may result in compensatory dilatation of a part or the whole of the lateral ventricles The air may be introduced at lumbar or cisternal puncture (air encephalography) or by ventricular puncture (ventriculography) an equivalent volume of cerebro spinal fluid must of course be withdrawn

(b) *Cerebral angiography*—X rays of the skull are taken immediately after the injection of a radio opaque solution into the internal carotid or vertebral arteries The injection is made percutaneously This method is useful in demonstrating congenital vascular abnormalities such as aneurysms and angiomas and vascular tumours Tumours may also be localized because they frequently cause displacement of the cerebral arteries

(c) *Myelography*—This is used in the diagnosis of spinal compression A radio opaque substance is injected into the sub arachnoid space either by lumbar or cisternal puncture after the removal of an equal volume of cerebro spinal fluid The substance which is heavier than spinal fluid is made to run up or down the spinal sub-arachnoid space by tilting the patient on a table under the X ray screen Partial or complete spinal obstructions can be demonstrated by this method

CHAPTER X

EXAMINATION OF THE EYE, EAR, THROAT AND NOSE

I THE EYE

NOTE first any obvious peculiarity about the eye Observe whether there is any undue prominence of one or both eyes Prominence of the eyes occurs in myopia in space occupying lesions in the orbit and in exophthalmic goitre It is associated in the latter disease with the presence of what is known as *von Graefe s sign* Ask the patient to look down If von Graefe s sign is present the upper lid lags behind the eyeball in its descent so that a portion of the sclerotic becomes visible This appearance of the eyes in exophthalmic goitre has two components First the eyeballs are displaced forwards (exophthalmos) This is thought to be due to the direct action of the thyrotropic hormone of the pituitary and is unaffected by thyroidectomy Secondly the eyelids are retracted This is due to overactivity of the cervical sympathetic and is abolished by thyroidectomy Occasionally in severe exophthalmos ocular palsy may supervene

Note also whether the blinking movements are increased or diminished in frequency Infrequency of blinking along with an increased size of the palpebral fissure constitutes *Stellwag s sign* of exophthalmic goitre

In paralysis of the cervical sympathetic the eyeball appears to recede and looks more sunken than normal (enophthalmos)

The occurrence of squint ptosis retraction of the upper lid and alterations in the pupil have already been considered The presence of any inflammation along the margins of the lids (marginal blepharitis) should be noted together with any fault in the apposition of the lid margins i e eversion (ectropion) or inversion (entropion) This can best be done by observing the set of the lashes

Next turn your attention to the conjunctiva It may be necessary to examine the conjunctiva lining the eyelids In order to do this in the case of the lower lid it is sufficient to depress the latter firmly

with the thumb To expose the inner surface of the upper lid make the patient look downwards place the right thumb at the upper part of the upper lid and push upwards so as to make the eyelashes stand out prominently Grasp the lashes between the forefinger and thumb of the other hand and evert the lid by rotating it round the thumb of the right hand Note the colour of the conjunctiva—whether it is pale injected or jaundiced and whether it is roughened velvety or smooth In conjunctivitis there is redness and congestion of the conjunctivæ with a mucoid or mucopurulent discharge

Look next at the cornea Note the presence of any ulceration or opacity Small opacities are described as *nebulæ* larger opacities are spoken of as *leucomata* Superficial opacities nearly always indicate active ulceration rarely does a superficial opacity mean a healed scar Look along the surface of the cornea and observe whether the light is reflected from it over the opaque spot or whether it is dull If the former is the case the opacity is deep-seated if the latter it is superficial Deep seated opacities are due to a previous keratitis often the result of a kerato-iritis peculiar to congenital syphilis

The term *arcus senilis* is applied to a crescentic opacity which is sometimes observed towards the margin of the cornea It usually appears at the lower part of the cornea first and then gradually extends round It occurs in old people and occasionally in healthy young folk or even adolescents (*arcus juvenilis*) It is without significance

It is often of importance to be able to say whether a patient is suffering from iritis or merely from conjunctivitis In each case the eye looks red and injected but the characters of the injection are different in the two conditions They are contrasted in the table on p 343

The tension of the eyeball should next be tested This is done by placing the two forefingers on the upper part of the sclera outside the upper lid while the patient looks downwards the other fingers resting on his forehead Then try for fluctuation The normal tension must be learnt by practice and any increase or diminution of it noted A diminished tension is found in a phthisical eye diabetic coma and severe dehydration from any cause A myopic eye also feels softer than normal An increased tension suggests glaucoma and contraindicates the use of atropine

Sympt m	C ; t vit	Int s
Pain	Discomfort burning it hing	Neuralgic pain
Photophobia	Absent	Present
Secretion	Mucoid or purulent discharge	Lachrymation (tears)
Impairment of vision	Absent or slight	Slight to considerable
S gn		
Redness	Maximal in fornices and cul de sacs minimal around corneæ	Ciliary or circumcorneal flush around l mbus colour maximal around *limbus minimal in* fornices
Cornea	Clear bright	Clear—but there may be small white specks behind the cornea (keratitis punctata)
Ant chamber	Clear	Hazy
Iris	Clearly patterned	Muddy
Pupil	Normal	Contracted, irregular
Secretion	Mucoid or purulent	Clear watery tears

The examination of the pupils (p 299) determination of the acuity of vision (p 279) and methods of examining the fields of vision (p 280) have been described in Chap IX Sometimes a patient wears glasses and it may be important to know what their refraction is To do this hold the glass in front of the eye and look at an object through it Then move the glass from side to side and watch the object If the latter seems to move in the opposite direction to the glass the latter is convex if in the same direction it is concave Patients with myopia use concave (divergent) lenses and those with hypermetropia convex (convergent) ones

The strength of the glass may be approximately determined by bringing the small lenses of the ophthalmoscope behind it until one finds that which abolishes the apparent movement of the object looked at

In order to tell whether the glass is spherical or cylindrical look at a straight object e g a window bar through the glass and then slowly twist the latter round If the glass is cylindrical the object looked at will appear to take up an oblique position Patients who are astigmatic use cylindrical glasses

The examination of the retina with an ophthalmoscope is an essential part of every medical examination Apart from the detection of local ophthalmic conditions valuable information may be obtained as to the state of the optic nerve head and of the arteries and veins of the retina

For a proper examination of the retina the pupils should be dilated by instilling a few drops of 2 per cent homatropine into the conjunctivæ This however may be dangerous in patients with glaucoma and is therefore contraindicated if the intra ocular tension appears raised It is also dangerous in elderly people with shallow anterior chambers Wide mydriasis may induce a con gestive attack under these conditions in an eye whose tension is normal One must therefore rely on the appearance as well as on the tension It is also contraindicated in patients with those acute intracranial lesions which require continuing observation of the state of the pupils e g suspected cases of epi or sub-dural hæmorrhage As soon as the examination is finished the effects of the homatropine should be counteracted by a few drops of 1 per cent eserine It is also helpful to examine the patient in a dark room In routine medical examinations it is usually possible with practice to examine the optic disc and a fair amount of the surrounding retina without resorting to mydriatics and in ordinary indoor light provided the eyes are shaded from any bright source of light Under these conditions however the periphery of the retina and the macular region are not well seen

The patient may be sitting upright or lying down whichever is convenient He is then directed to look straight ahead and if possible to fix his eye on some distant object He should in any case be asked to keep his eyes still and go on looking straight ahead even if the observer s head gets into his line of vision The observer then directs the light of the ophthalmoscope into the patient s eye and looks through it The so-called red reflex will be seen at once unless the retina is obscured by corneal lenticular or vitreous opacities These can often be defined by using the plus lenses of the ophthalmoscope and in particular lenticular opacities or

cataracts should be examined for with the +12 lens They are seen in various forms early ones often appearing at the periphery of the lens and spreading inwards like the spokes of a wheel towards the centre

The observer should learn to relax his own accommodation by imagining that he is looking at a distant object, and should start the examination with a +8 or +10 lens in the aperture of the ophthalmoscope This makes certain of not missing opacities of the media. The ophthalmoscope should then be brought *as close as possible to the patient s eye* and the light directed over the cornea and media. Gradually reduce the lenses to zero and direct the light a little inwards from the axis of the eye on to the optic disc If it is shone directly through the pupil in the axis of the eye it will be directed on to the macula, and the pupil if not dilated with mydriatics will contract and make it difficult to see the fundus at all. When the retina is seen the details may or may not be in focus If they are not, the graduated lenses of the ophthalmoscope must be tried in turn, until one is found which brings the retina into sharp focus If the disc is not seen, it is only necessary to follow a vessel back to its point of emergence

Provided the observer's eye is emmetropic and his accommodation properly relaxed the strength of the lens necessary provides a rough indication of the degree of refractive error in the patient s eye in dioptres Plus lenses indicate hypermetropia, and minus ones myopia.

The optic disc the blood vessels the macular region and the periphery of the fundus must be studied in detail in each case

1 The optic disc —Note —

(1) Its shape Normally this is circular Sometimes it is oval If there is astigmatism present, the disc will appear to be oval, although it is really circular This apparent oval shape may be distinguished from that which is real by moving the lens backwards and forwards If the disc is really oval it remains unaltered if it is only apparently oval its shape will be found to vary with the position of the lens

(2) Its colour The normal disc is of a rosy tint but distinctly paler than the rest of the fundus The nasal side is normally rather redder than the other

In atrophy of the optic nerve the disc becomes very pale and may

even be dead white or greyish in tint In active hyperæmia of the disc its colour approaches in intensity to that of the rest of the fundus Such active hyperæmia is often present in high degrees of hypermetropia In passive hyperæmia of the disc the veins are alone affected and the general tint is not altered

(3) The presence or absence of a physiological cup (*see* Frontispiece) and its size Do not mistake the pallor produced by a very large cup for the pallor of optic atrophy

(4) The edge of the disc It should be clear and well defined—especially at its outer side As the vessels run across it they should not be observed to tumble over at all This tumbling over if present is best evinced by the sudden disappearance of the central light stripe on the vessel

(5) The surroundings of the disc This part of the fundus should be carefully searched if one is looking for hæmorrhages or miliary tubercles as both of these are more often encountered in the immediate neighbourhood of the disc than at other parts of the fundus

2 **The blood vessels** —The arteries are normally distinguished from the veins by the following characters They are only two thirds to three quarters the breadth of the veins and they are not so dark in colour They have a broader better defined and more continuous light stripe along their centres Normally the arteries do not pulsate They may be observed to do so in cases of aortic regurgitation and in increased intraocular tension The veins sometimes pulsate even in normal eyes A point where an artery crosses a vein should be chosen for observation In health it should be possible to see the vein walls through the artery neither artery nor vein should be altered in direction at such a crossing and neither should the calibre of the vein be reduced

3 **The macular region** is situated about two discs breadth from the outer edge of the disc It is recognized by being rather darker in colour than the rest of the fundus by being devoid of blood vessels and frequently by being surrounded with a halo of reflected light, producing a shot silk appearance The macula itself is in the centre of the region and is rather pale in colour and often glitters somewhat Changes in the macular region are important in that they interfere more with vision than similar changes in any other part of the fundus In cases of hypertensive retinopathy a

circle of white spots may often be observed arranged around the macula (*see* Frontispiece)

4 **Periphery** —Inspection of the periphery of the retina is important as it is here that some changes—such for example as diabetic retinopathy and retinitis pigmentosa—are first to be detected

The following is a brief description of the chief changes met with in the fundus which are of importance from a medical point of view —

Papillœdema was at one time known as optic neuritis It arises as a result of pressure and not of inflammation It is usually bilateral and begins as a mere passive congestion of the disc with slight œdema At this stage the veins are fuller than normal the optic cup may be obscured and the margins of the disc are slightly blurred The change in the edge of the disc usually begins at its upper and lower nasal margins These parts should therefore always be most carefully inspected

As the process progresses the œdema increases and the disc becomes definitely swollen (*see* Frontispiece) This is best recognized by the fact that one requires (provided accommodation is fully relaxed) the aid of a convex lens behind the mirror in order to bring the vessels on the disc clearly into focus The veins are still larger than before and distinctly tortuous Pathological tortuosity of the veins occurs at right angles to the plane of the retina Tortuosity in the same plane as the retina may be quite normal Often the veins can be observed to tumble as it were over the edge of the swollen disc The arteries are smaller than normal and may be partly obscured by the presence of exudation The edge of the disc is no longer clear even on indirect examination but fades off into the surrounding retina Small hæmorrhages may be observed on or near the disc

In cases of raised intracranial pressure intense œdema of the optic disc may result in extreme swelling a condition known as choked disc an expression that well describes the condition seen The veins are distended and tortuous and here and there the path of the vein is hidden by exudation and hæmorrhages occur on the surface and margins of the swollen and distorted disc

It is often important to decide whether papillœdema is advancing or not This can only be done if the disc has been examined on a

previous occasion The best *criterion* is the degree of *swelling* of the disc In order to estimate this be sure that your own accommodation is thoroughly relaxed Notice first whether the retina can be seen quite clearly without the aid of a lens If the eye is emmetropic one ought to be able to do so If the refraction is abnormal place behind the *mirror the strongest + or the weakest −* lens which is required to bring the vessels on the retina clearly into focus Then look at the vessels on the disc Owing to the swelling the vessels are nearer the observer s eye than they should be and a + lens must therefore be brought behind the mirror in order to *enable one to focus them clearly* The *strength of the lens required* is the measure of the amount of swelling which is present in the disc Suppose for example that one requires to use +1 D in order to focus the retina clearly (i e the patient has 1 D of hypermetropia) but that in order to focus the vessels on the disc one requires to make use of a +6 D then there is obviously +5 D of swelling *Roughly speaking every 3 D = 1 mm of swelling* In this way one can estimate the amount of swelling from day to day and so determine whether the condition is advancing or receding

Papillœdema may occur in any condition in which the intracranial pressure is raised It is thus seen in cases of cerebral tumour though less frequently than in the past when ancillary methods of diagnosis were lacking Papillœdema may follow subarachnoid hæmorrhage but then it is usually slight Again the papillœdema which may accompany cerebral abscess or sub-dural hæmatoma is generally of minor degree Patients with acute meningitis rarely have papillœdema but it may be encountered in sub-acute or chronic forms of meningitis Papillœdema does not *occur in cerebral hæmorrhage or thrombosis but it may follow* thrombosis of the cerebral veins or sinuses

Unilateral optic atrophy due to direct pressure on the optic nerve associated with contralateral papillœdema due to intracranial hypertension is seen particularly with meningiomas of the medial part of the sphenoidal ridge (Foster Kennedy syndrome)

Swelling of the optic disc is not infrequently seen in other than intracranial diseases especially in chronic nephritis and malignant hypertension

Particular attention should be paid to the condition of the blood vessels as this may not infrequently help in deciding the cause of

the swelling of the optic disc Examine carefully the crossings of arteries and veins (*see* p 346)

Papillitis (intra-ocular optic neuritis) is an inflammatory swelling of the optic disc which occurs as part of a neuro retinitis It should not be confused with papillœdema for the pathology of the two conditions is quite different although the ophthalmoscopic appearances may be very similar Generally speaking in the optic neuritis of a neuro retinitis the swelling is moderate —2 to 3 D— gradually fading into the surrounding retina which may show signs of inflammation (exudates hæmorrhages vitreous haze and so on) The distension of the veins is less marked than in papillœdema Distinction of papillœdema from neuritis is made largely on the absence in the former and presence in the latter of a field defect (usually central scotoma)

In those cases in which the optic neuritis is limited to the retro bulbar part of the nerve there is no visible change in the nerve head in the acute stage but optic atrophy may supervene

Optic atrophy —The most striking change in the fundus in this condition is the pallor of the disc and the smallness of the arteries on it The atrophy may be primary secondary or consecutive It is not always easy to say from a mere inspection of the fundus which variety it is that one has to deal with and the longer the process has gone on the more difficult does the diagnosis become

(*a*) **Primary or simple atrophy** —The thinning of the nerve fibres renders very visible the structure of the lamina cribrosa and the disc acquires a mottled appearance (*see* Frontispiece) The disc becomes dead white in colour and its margins are unusually distinct

(*b*) **Secondary or post neuritic atrophy** —Here the atrophy is secondary to papillœdema The edges of the disc are indistinct and white streaks can be seen radiating along the vessels into the retina

(*c*) **Consecutive atrophy** —Here the atrophy is consecutive to changes in the retina the disc looks like a bit of dirty parchment and pigmentary changes will be seen in the retina

Retinal hæmorrhages may be observed in hypertension chronic nephritis diabetic retinopathy aplastic anæmia thrombocytopenic purpura and in any condition in which emboli lodge in the retinal

arteries or where the retinal circulation is embarrassed When superficial the hæmorrhages are elongated with so-called flame shaped edges When deep they occur as dark red blotches or as minute discrete rounded spots Subhyaloid hæmorrhages are occasionally seen as very large rounded hæmorrhages with a straight horizontal upper border they sometimes accompany sub-arachnoid hæmorrhage

Embolism of the central artery of the retina —The appearance of the fundus is characteristic Look at the macular region for a peculiar round cherry red spot The whole or part of the disc is pale and its arteries are empty The retina as a whole is somewhat milky looking from the presence of œdema

Thrombosis of the central retinal vein —There is intense swelling of the optic disc with gross venous distension and radial hæmorrhages

Retinal arterio sclerosis occurs either as part of generalized decrescent arterio-sclerosis or as a complication of benign hypertension The principal changes are —(1) Tortuosity and irregularity in the lumen of the arteries (2) nipping indentation or deflection of the veins where they are crossed by the arteries (3) small flame shaped hæmorrhages and (4) small sharply-defined white patches of exudate in the region of the macula

Hypertensive retinopathy —This condition met with in some cases of chronic nephritis was originally called albuminuric retinitis However it is not found except in the presence of high blood pressure and therefore it is more appropriate to call it hypertensive The changes consist in (1) the presence of papillœdema with marked fullness of the veins (2) the occurrence of hæmorrhages on or near the disc (3) the development of white shining spots around the disc at a distance of about three discs breadth from it and of similar but much smaller spots arranged in a stellate form around the macular region (*see* Frontispiece) and (4) retinal arterio-sclerosis Any one of these changes may be present without the others

Diabetic retinopathy may bear a close resemblance to hypertensive retinopathy but distinguishing characteristics are often present

The fundamental change is the formation of capillary micro-aneurysms which may be seen as sharply-defined dark red spots The hæmorrhages tend to occur in the deeper layers of the retina and are therefore punctate or blot like The white masses of exudate are discrete and waxy and there is no star figure at the macula Retinal arterio-sclerosis is commonly present.

In leukæmic retinopathy the appearances vary The red reflex and blood vessels are pale the veins enlarged and round white spots up to 2 mm diameter occur sometimes fringed with blood Hæmorrhages of various kinds are common

Disseminated choroiditis —This is frequently an important sign of previous syphilis There are small white patches of various shapes and sizes surrounded by heaped up black pigment (*see* Frontispiece) That the lesions are situated in the choroid can be recognized by the fact that the retinal vessels pass over them

Choroidal miliary tubercles may be looked for in cases of suspected acute miliary tuberculosis and tuberculous meningitis They will rarely be recognized as ill-defined rounded dull yellow lesions usually about half a disc's breadth in diameter They are usually of little diagnostic importance

Opaque or medullated nerve fibres occur in the form of one or more broad streaks of brilliant white radiating for a short distance from the disc and showing a characteristic feathered edge (*see* Frontispiece) The condition is a harmless congenital abnormality

II THE EAR

Note any unusual appearance of the external ear Compare one ear with the other Movement of the pinna does not normally give rise to pain Note any displacement or swelling The skin should be intact and healthy make a note of any redness fissuring or desquamation At the entrance to the meatus there should be fine hairs more obvious in the male than in the female If there is any bleeding or discharge from the meatus make a note of it

Examine next the inside of the meatus which is a skin lined passage just over 2 5 cm (1 in) in length and only about 9

in its greatest diameter so that it will not admit even the little finger It slopes upwards and forwards and ends in the tympanic membrane In order to obtain a well lighted and magnified view of this passage it is necessary to use an electric auriscope

The auriscope consists of a metal cylinder containing a small dry battery At one end of the instrument is a lens through which the examiner looks down a funnel shaped speculum which is lighted by a small electric bulb

The narrow end of the speculum is introduced into the meatus (Fig 79) The examiner should sit or stand with his eye on a level

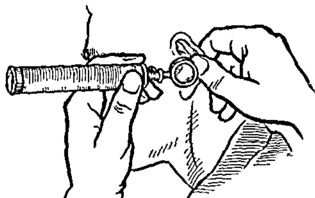


Fig 79 —The auriscope in use

with the patient s ear If the patient is recumbent in bed the right ear should be examined from the right side of the bed and the left ear from the left side of the bed with the patient s face turned away from the examiner To examine the right ear the auriscope is held in the right hand and for the left ear it is held in the left hand The free hand holds the pinna of the ear between the thumb and first two fingers and resting the little finger against the mastoid bone behind pulls the pinna gently outwards from the skull in order to straighten out the meatus This makes it much easier to see the tympanic membrane The outer third of the meatus is *movable and cartilaginous the inner two-thirds is bony and* immovable

Note that the little finger of each hand rests on the cheeks and

skull respectively These positions keep the instrument steady and prevent injury to the ear should the patient suddenly turn his head Introduce the speculum gently The opening of the meatus is oval and the speculum is usually made oval to fit it Each auriscope is provided with three specula and it is helpful to use the largest one that will fit The speculum should lie gently in *the meatus just beyond the hair line The auditory meatus is very* sensitive and protected at its opening with hairs sebaceous glands and apocrine ceruminous glands but there are no hairs or glands in the skin of the inner two thirds of the meatus The examiner should look upwards and in a forward direction and should not expect to see the whole tympanic membrane in one view The lens of the auriscope may be moved slightly but the end of the speculum in the meatus should be kept as still as possible until the whole of the tympanic membrane and meatus has been inspected A gentle examiner will see much more than one who is inconsiderate and it is as well to remember that an ear should be treated with as much respect as an eye

The tympanic membrane (Fig 80) lies at the end of the meatus It is covered by skin and is so thin and translucent that the shadow of the cavity of the middle ear behind gives it a faint bluish grey tinge Near the centre of the tympanic membrane is the handle of the malleus and it is convenient for the examiner to pick out the malleus first in his view down the auriscope At the upper end of the handle of the malleus is a nipple like protrusion This is the lateral process of the malleus and is very useful as a landmark in case of difficulty In front and behind this lateral process are the anterior and posterior ligaments which should stand out clearly That part of the tympanic membrane which lies below these ligaments is called the membrana tensa The membrana flaccida lies above these ligaments

Deep to the membrana tensa is the bony cavity of the middle ear Deep to the membrana flaccida is the epitympanic recess of the middle ear which lies just under the tempero sphenoidal lobe of the brain (Fig 80) These important anatomical relationships of the middle ear are seen in the diagram

It is important to describe *abnormal changes in the tympanic membrane* such as retraction of the handle of the malleus or loss of translucency of the membrane Œdema and redness due to

inflammation in the middle ear may cause obliteration of the normal landmarks In very recent and acute otitis media the tympanic membrane is bright red and swollen but as œdema increases and obstructs the venous return the membrane may become cyanotic

Any visible *perforation* in the tympanic membrane should be

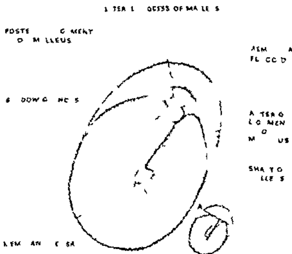


Fig 80 —Diagram of the right Tympanic Membrane

In the small diagram A shows the point where the tympanic antrum opens into the middle ear and B shows where the eustachian tube opens into the middle ear

described as lying anterior to posterior to or above or below the malleus

If the tympanic membrane has ruptured recently a thin blood stained discharge may be seen coming from an ill-defined perforation An old perforation is more easily seen its edges are smooth and there is usually no bleeding, but there is sometimes a purulent discharge

A large perforation or complete destruction of the tympanic membrane may expose the medial wall of the middle ear which is usually pink and moist but may be reddened by inflammation

On the medial wall lies the tympanic plexus of nerves and the oval and round windows of the inner ear Changes in temperature or barometric pressure are liable to cause symptoms of pain and giddiness in cases where the middle ear is widely exposed and such ears with perforations are obviously more liable to infection from the outside

A reddish glistening movable and painless swelling protruding from a perforation in the tympanic membrane suggests a **polypus** A dull but intact tympanic membrane accompanied by des quammation of the skin of the external auditory meatus with a moist or purulent exudate is seen in **dermatitis** of the meatus A swelling in the meatus if painful suggests a furuncle A rounded swelling on the wall of the meatus which gives no pain and is hard and covered with sensitive skin suggests an osteoma of the meatus

Any œdematous swelling behind the pinna should be noted It may indicate a cellulitis if a furuncle is present in the meatus it may be evidence of periostitis and **mastoiditis** if otitis media is also present

Because the middle ear cleft lies within the bone of the base of the skull any patient with a history of chronic purulent otitis media may at some time develop **intracranial complications** and this should be borne in mind during clinical examination

Dilatation of the blood vessels running down the malleus of an intact tympanic membrane occurs after slight **trauma** Any tear in the tympanic membrane or presence of fresh blood in the meatus after accidents involving fracture of the base of the skull or expos ure to blast injury from explosions should be particularly noted Pain following rapid descent in an aeroplane may be accompanied by bleeding from the tympanic membrane

SYRINGEING THE EAR

If the meatus is blocked with cerumen or contains a foreign body it may not be possible to see the tympanic membrane until the obstruction has been cleared **Wax** is often coloured dark brown by concentration of its pigment It may be mixed with desquamated epithelium and hairs and is sometimes firmly wedged and very hard It is best to soften it by instilling olive-oil drops frequently for several days The patient s head is p

on one side The olive oil need not be warmed by artificial means but if the weather is cold the oil may be dropped first into the shell or concha of the ear where it will be warmed quite satisfactorily by the skin to body temperature The pinna can then be raised and the oil will run from the concha into the meatus

When the wax has been adequately softened the mass can be removed by gentle syringeing with tap water warmed to body temperature The stream of water from the syringe should be directed at a point between the mass of wax and the meatal wall The pinna is held out from the head whilst syringeing in order to straighten out the meatus

In order to catch the return flow of water coming out of the meatus and to avoid wetting the patient s clothes a waterproof cloth should be placed in position close to the patient s neck and the patient himself should hold a receiver under the ear

The syringes most commonly used for this purpose are the metal one handed aural syringe or the rubber Higginson type syringe A good light is essential and great care should be taken to make sure the water is neither too hot nor too cold If it is too hot it will cause pain and possibly serious and permanent damage and if it is too cold it will cause vertigo It should feel pleasantly warm

Before syringeing any ear the meatus should be examined with the auriscope On no account should an ear be syringed if there has been recent pain accompanied by a recent upper respiratory infection These symptoms suggest an inflammation of the middle ear especially if loss of hearing is present as well and syringeing over an inflamed and œdematous tympanic membrane may cause further damage and pain Equally it may be very unwise to syringe out an ear if a perforation in the tympanic membrane is known to be present or is suspected

After syringeing an ear the meatus should always be inspected again with the auriscope If the obstructing wax has been removed the meatus should be mopped quite dry with thin twists of cotton wool because moisture nourishes bacteria on skin After inspection of the tympanic membrane a few drops of olive oil should be instilled into the meatus It is most dangerous to syringe an ear if blood is escaping from the meatus after a fracture of the base of the skull

Various solvents for dissolving hard wax have been suggested from time to time They dissolve the wax but must be used with

great caution because they may penetrate and damage the ducts of the ceruminous and sebaceous glands For whereas it may be necessary to remove a mass of hard pigmented wax from a meatus in order to inspect the tympanic membrane it is also important to prevent further damage to the ceruminous and sebaceous glands A thin layer of normal cerumen mixed with sebum is bactericidal and very necessary for the health and protection of the meatal skin

Cleaning the meatus of the ear —If the meatus is filled with pus or discharge a culture should be taken and the sensitivity of the organisms determined Then in order to see the tympanic membrane the discharge must be gently mopped out with thin twists of cotton wool on thin wooden applicators This is important because if the cotton wool twists are thick and fill the meatal opening they will act as pistons and force the discharge back through a possible perforation It is helpful to dip each cotton wool twist in olive oil just before use because this fixes the discharge and makes the mopping out operation more comfortable The used twists of cotton wool contaminated with discharge should be burned

The cotton wool twists are made by the examiner A pinch of cotton wool is teased out flat and twisted neatly and firmly round the end of a thin wooden applicator so that about half an inch of firm cotton wool twist is left extending beyond the wood This cotton wool extension should be about as thin as the diameter of the wooden applicator The applicator can now be used quite safely in the meatus of the ear and because the end of the cotton wool that may reasonably be expected to come into contact with the tympanic membrane is always left soft and fluffy no harm should come to that delicate structure The extension easily conforms to the shape of the meatus without injury to its delicate skin and the examiner can make his task much easier by gently pulling on the pinna to straighten out the passage The tympanic membrane is just over one inch from the bottom of the concha

The skin of the posterior and inferior walls of the meatus of the ear is supplied by the **vagus nerve** The well known connections of this nerve account for reflex coughing or fainting if this skin is irritated Vomiting may follow the syringeing of children s ears and occasionally heart failure may be similarly induced in elderly people

III THE LARYNX

Examination of the larynx gives information about the adequacy of the respiratory airway and the causes of changes in the voice

To perform laryngoscopy it is necessary to use a *laryngeal mirror* This is a rounded mirror set at an angle at one end of a long thin metal handle (Fig 81) The idea is to hold the back of the mirror lightly but firmly against the soft palate so that an image of the vocal cords will appear in the mirror

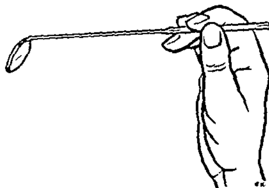


Fig 81 —Holding the laryngeal mirror

The examiner seats himself facing the patient He will have to sit very close to his patient with his legs to the side nearest to the source of light A *standard lamp* with a good bright beam is placed at the side of the patient on a level with the patient s mouth and its beam is directed at the examiner For a right handed examiner the light is placed at the patient s left The examiner must wear a *face mask* to protect himself should the patient cough directly at him during the examination most patients with serious lesions of the larynx are very helpful and co-operative but anxious patients with slight diffuse inflammation of the larynx may be very sensitive and cough easily Next the examiner places a *head mirror* on his forehead over his right eye A head mirror is concave and has a hole in the centre through which the examiner s right eye will inspect the image in the laryngeal mirror The mirror is fastened

to the head by a band and can be adjusted easily because it is fitted with a ball and socket joint The beam of light from the standard lamp is reflected on to this concave head mirror then on to the laryngeal mirror and so down to the larynx The examiner s eye looks directly down the centre of this reflected light beam and so the lighted image in the laryngeal mirror becomes clearly visible

First the patient s mouth is inspected and if artificial teeth are present his *dental plates should be removed* The laryngeal mirror is *warmed* by placing it in hot water or holding it for a second over a spirit flame This is to prevent misting of the mirror when the patient exhales The mirror is wiped clean and tested to make sure it is not too hot by holding it firmly against the sensitive ventral surface of the examiner s wrist

The patient is asked to lean a little forward and to open his mouth and put out his tongue With a small square of clean linen in the left hand the examiner takes hold of the tip of the patient s tongue between his thumb and second finger The linen (or gauze) square is necessary or the fingers will slip on the moist tongue The index finger of the same hand gently lifts the upper lip

The light from the head mirror is directed at the patient s mouth The laryngeal mirror is held in the right hand (Fig 81) and very carefully in order to avoid touching the tongue the small round mirror is placed deliberately and steadily against the soft palate near the base of the uvula The shaft of light from the examiner s head mirror should now shine directly on to the laryngeal mirror and the laryngeal mirror is gently adjusted until the vocal cords come into view

Laryngoscopy requires no local analgesic in the great majority of examinations but it does require the confidence of the patient in the examiner and the examiner s confidence in himself The examiner must train himself to make rapid and accurate observations

To facilitate matters the patient is asked to continue breathing throughout the examination If the patient is concentrating on breathing he will not think of retching or coughing If movement of the cords is required the patient is asked to say E

The larynx should be inspected with great care (Fig 82) The epiglottis lies in front at the base of the tongue Normally its upper curved edge is clearly seen its colour is a pale yellow Any abnormal position should be noted The base of the tongue and

the valleculæ are next examined Both piriform fossæ are inspected and the presence of excessive froth or mucus round the œsophageal opening behind the larynx or in either piriform fossa should suggest upper œsophageal obstruction or paralysis In upper œsophageal obstruction the saliva and mucus from the mouth and

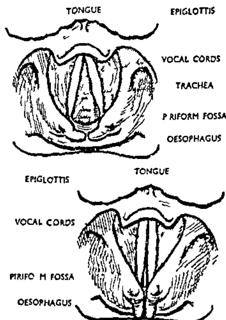


Fig 82 —The larynx as seen in the laryngeal mirror

upper respiratory tract cannot get away easily and may overflow into the larynx explaining the patient s irritating cough

The vocal cords or vocal folds as they are now sometimes called are normally clear cut and of a pale yellow glistening appearance The rest of the laryngeal mucosa is normally a moist pinkish red colour Between the vocal cords is seen the lumen of the trachea and the outlines of the tracheal rings The presence of any pus or mucus in the trachea and any narrowing of the tracheal inlet should be noted

Displacement of the larynx suggests inflammation or new growth in the tissues closely adjacent to the larynx

The **epithelium** should be inspected for any irregularity ulceration change of colour or presence of œdema Raised irregular epithelium suggests malignant changes Ulceration with œdema makes one think of tuberculosis if there is evidence of pulmonary infection Œdema alone indicates inflammation or trauma or both Diffuse redness of sudden onset suggests acute laryngitis

The **movement** of the larynx should be noted The movement of both sides of the larynx should be symmetrical When normal movement is absent the examiner must ask himself whether some local condition such as fixation due to malignant changes or myopathy is preventing normal movement or whether some lesion along the course of the recurrent laryngeal nerves is responsible If loss of movement is caused by some local change there will usually be signs of swelling or obstruction or ulceration or there may be a past history of poliomyelitis or diphtheria

If the larynx looks quite normal except for the paralysis common sites along the courses of the recurrent laryngeal nerves worthy of clinical examination are —(1) the thyroid gland (2) the apices of the lungs and structures related to the under surface of the aorta and (3) the cervical œsophagus

Absence of movement of one or both cords should be carefully noted If one arytenoid lies a little in front of the other this is often suggestive of unilateral paralysis and may be helpful if the larynx is difficult to examine

In **unilateral paralysis** the examiner is struck by the overaction and compensation of the normal cord which comes right across the middle line to meet the paralysed cord if the patient endeavours to say the vowel E It is this remarkable *compensation* which explains the apparently normal voice in unilateral paralysis and accounts for the tiredness of the voice towards the end of the day There is usually no stridor and no apparent obstruction to the airway Compensation is rapid Some very temporary difficulty with swallowing is experienced due to unilateral paralysis of the upper sphincter of the œsophagus (crico pharyngeus) which is also supplied by the recurrent laryngeal nerve

The extraordinary compensatory powers of the larynx are well seen in **bilateral paralysis** where although the voice is undoubtedly weak it is remarkably clear those muscles not supplied by the

recurrent nerves taking on added functions Even when the complete larynx has had to be removed because of malignant changes and a permanent tracheotomy is the only respiratory passage many patients develop an amazingly good voice Folds in the pharyngeal walls are used for phonation and the relaxed oesophagus makes an efficient air reservoir

In unilateral paralysis the patient does not complain of shortness of breath on exertion whereas in bilateral paralysis there is great narrowing of the laryngeal airway because the main abductors of the cords are paralysed and the patient does complain of real shortness of breath on exertion There is usually acute distress if both cords are paralysed suddenly but with reassurance and when adaptation is established most patients complain of little difficulty in breathing when at rest or during light movement The stridor during sleep is very inconvenient for others There is no stridor in unilateral paralysis It must be remembered that recovery does take place more often than is usually realized in both unilateral and bilateral paralysis of the recurrent laryngeal nerves

It is very important to examine the larynx in any case of hoarseness that has persisted for two or three weeks Intermittent attacks of hoarseness or loss of voice with a complete return to normal between attacks should suggest simple laryngitis or hysteria If any growth is present on either vocal cord the voice will never clear until the growth has been removed Whenever the epithelium of the larynx is ulcerated or irregular an X ray of the lung fields and the Wasserman reaction should be done before any question of biopsy is considered If there is purulent sputum this also should be examined

When considering the advisability of biopsy as an aid to diagnosis it must be remembered that interference with the edge of a growth (where the normal tissue cells of the body are doing their best to resist the encroachment of malignant cells) may cause rapid extension of the growth in the larynx Thus is particularly important when a growth is small and a complete cure is probable In such cases the best and safest diagnostic biopsy is often the removal of the complete tumour surrounded by an adequate margin of healthy tissue

No laryngoscopy is complete without an examination of the neck The position of the thyroid cartilage and trachea, should be inspected and any displacement noted Movement of the

larynx on swallowing should be free and any unusual lump or enlarged nodes must be felt for

IV THE NASO PHARYNX

Examination of the naso pharynx is carried out in much the same way as that described above for laryngoscopy and with the same precautions only here the mirror is smaller and its handle is bent to avoid touching the tongue The tongue may be depressed with a spatula and the small mirror is placed just behind the soft palate facing upwards The examination requires considerable skill and confidence and as with laryngoscopy some patients are easy to examine and some very difficult indeed

The posterior nares lying to either side of the sharply defined vomer come into view in the mirror and the posterior ends of the inferior and middle conchæ can be seen To either side lie the openings of the eustachian tubes The presence of any tumour or pus or change in the normally pink moist epithelium should be noted

V THE NOSE

The outside of the nose must be inspected first Old scars may indicate past trauma Recent cuts or bruising should be noted Redness or swelling may be caused by inflammation Displacement of the nasal bones to right or left or depression of the bridge suggests trauma If the bones are firm and painless the injury will not have been of recent origin Collapse of the bony or cartilaginous bridge may have followed necrosis With a finger and thumb on the outside of each nostril the anterior part of the cartilaginous septum can be palpated and any gross displacement or thickening of the septum detected

The skin round the nostrils must be inspected for fissuring or redness The presence of discharge whether blood stained or mucopurulent or watery must be noted and whether the discharge is confined to one nostril only or comes from both.

To inspect the vestibule of the nose and the nasal passages a head mirror is needed There are several advantages in using a concave head mirror for examinations of the nose naso pharynx and larynx The examiner can look right down the centre of the beam of light

which his head mirror reflects into these narrow and darkened passages Both hands of the examiner are free and his eyes are shaded Other forms of light such as torches or head lamps or lighted specula can be tried but they are not as satisfactory as the perforated head mirror with reflected light

The vestibule of a small child s nose and the openings of its nasal *passages can be inspected by tilting the nose upwards with a finger*

To examine an adult s nose properly a *nasal speculum* is needed The speculum is held in the left hand (Fig 83) and the light beam reflected from the head mirror is directed on to the patient s nostril

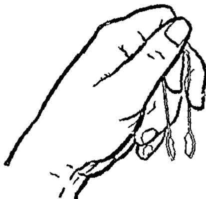


Fig 83 —*Holding the nasal speculum*
Figs. 79–83 drawn b M Ch les A Keogh

The speculum is closed and gently introduced into the nostril *The spring is allowed to open a little but never to its fullest extent* Gentleness is all important

The skin of the vestibule extends inwards for about half an inch and it is well supplied with protective hairs particularly in males The skin of the nasal vestibule is heir to all the common skin diseases There is a clear line where the pale skin meets the pink moist mucous membrane of the nasal passages Medially lies the nasal septum which is commonly displaced by trauma A hæma toma of the nasal septum causes a swelling which is soft to the touch unlike the normal septum which is firm Note the presence of any ulceration or perforation of the septum or the presence of

any bleeding point Laterally is seen the **inferior nasal concha** This should be moist and pink and as its epithelium can become pale and greatly thickened in hay fever and asthma comparison should be made with the normal Above the inferior concha can be seen the anterior part of the **middle concha** The presence of pus coming from under the middle concha strongly suggests purulent sinusitis for most of the accessory nasal sinuses open under this concha Nasal polypi appear as moist greyish swellings occluding the air passages They are easily movable and painless unlike the conchæ which are fixed and tender Hyperæmia of the mucosa and the presence of muco pus in the nasal passages probably indicates infection A persisting one sided blood stained nasal dis charge may indicate the presence of a foreign body or malignancy if present in an older person The presence of any raised irregular or bleeding epithelium should be noted

The nasal passages form part of the upper respiratory tract and should never be examined without reference to the lower respiratory tract All air reaching the lungs has to pass through the nasal passages to be filtered warmed and moistened and any infection or obstruction in these passages may ultimately have some effect on the health and function of the lungs and bronchi The presence of nasal obstruction must be noted In children mouth breathing due to nasal obstruction gives rise to a change in the pattern of facial expression which is indicated by the open mouth and sagging of the facial muscles The lips and mouth are dry

Severe nasal obstruction may prevent air reaching the olfactory area and so cause loss of the sense of smell

VI THE NASAL SINUSES

To examine the **maxillary** sinuses —

(1) Palpate the bony walls of the sinus giving particular attention to the bone under the eye Compare the outline on the two sides Any tenderness swelling expansion or depression of bone must be observed The palate and alveoli must also be inspected and palpated from inside the mouth

(2) Examine the nasal passages to detect any evidence of pus or polypi appearing from any of the normal openings of the sinuses

(3) **Transillumnate** the sinuses This is done by placing a strong light in the centre of the hard palate The patient closes the lips

round the handle of the transilluminating lamp The examination should be performed in a dark room or with curtains drawn round the bed Light from inside the mouth shines through the hollow sinuses producing a light in the bone under each eye and a reddish reflection from the retinæ

Comparison between the two sides is made Pus in the maxillary sinus throws a dark shadow Patients will differ greatly in the thickness of their maxillary bones Some transilluminate clearly others with difficulty Transillumination is a useful clinical aid because if the sinuses are perfectly clear on transillumination an X ray may be unnecessary

If any abnormal shadow is found an X ray of the sinuses will give additional information and will help to confirm the clinical examination

To transilluminate the *frontal sinuses* the light is placed under the frontal ridge of the orbit The *ethmoid* and *sphenoid* sinuses are best examined by X ray combined with clinical inspection of the nasal passages

CHAPTER XI

LOCOMOTORY SYSTEM (BONES, JOINTS GAIT)

THE locomotory system includes the muscles bones and joints The examination of the muscles is most conveniently considered along with that of the nervous system (Chap IX) There remain for consideration the bones and joints

1 THE BONES

In examining the **long bones** of the limbs look for any alterations in shape or outline for localized swellings in the bone for signs of fracture and for evidence of undue tenderness Alteration in the shape of the bones occurs particularly in rickets (Plate 24) In osteitis deformans the bones are both deformed and enlarged (Plate 25) Localized swellings are mostly due to surgical conditions Spontaneous fractures may occasionally assist in the diagnosis of carcinoma generalized osteitis fibrosa (hyperparathyroidism) osteogenesis imperfecta or multiple myeloma Undue tenderness of the bones apart from surgical conditions is found in generalized osteitis fibrosa myelomatosis occasionally in carcinomatosis of bones and very rarely in leukæmia

The vertebral column and skull demand special attention Note the presence of any local projections or angular deformity of the vertebral spines and state which vertebræ are involved and at what level the projection is most prominent Landmarks are C 7 (vertebra prominens) and the last rib articulating with the 12th thoracic vertebra In many cases however the last rib cannot be distinctly felt and is therefore rather untrustworthy as a guide

Note also any curvature of the spinal column as a whole or of part of it distinguishing carefully such general curvature from the local projections above referred to

The curvature may be in an anterior posterior or lateral direction Anterior curvature is termed *lordosis* and is commonest in the lumbar region General posterior curvature is spoken of as

kyphosis It occurs most typically in the thoracic region in old persons and must be distinguished from the localized angular deformity of spinal caries Lateral curvature is termed *scoliosis* and may be towards either the right or the left side It is always accompanied by a rotation of the bodies of the vertebræ in such a way that the spines come to point towards the concavity of the curve

Ask the patient to stoop down notice the degree of mobility of the vertebral column and the occurrence of any pain during stooping noting the exact site of the latter if present Then pass the hand down the vertebral column and observe whether any tender spots can be discovered To elicit more deep-seated tenderness of the vertebræ it may be necessary to punch the spines gently with the fist from above downwards observing the point at which the patient complains of pain and verifying the observation by repeating the process from below upwards

In studying the skull note its shape

Is it of the dolichocephalic (long headed) or the brachycephalic (bullet headed) type? Certain well recognized types of abnormal skull are met with In *hydrocephalus* the skull tends to assume a globular form The forehead is overhanging and the eyes are pushed down so that the upper part of the sclerotic is exposed The lateral aspects of the skull (above the ears) project outwards If the patient is a child as is usually the case the fontanelle is wide and bulging and often fluctuates very distinctly The sutures may be opened up and imperfectly ossified areas (*craniotobes*) may be detected in the bones In *rickets* the skull tends to be square or oblong and box shaped The frontal and parietal bones often show central thickening (bossing) The forehead however does not overhang nor are the eyes depressed and although the fontanelle is usually widely open it doe not bulge as it does in hydrocephalus nor are the sutures opened up In *congenital syphilis* the forehead is vertical the frontal eminences are often exaggerated and the bridge of the nose is depressed In *acromegaly* the supra-orbital ridges and bones of the face particularly the lower jaw are enlarged so that in relation to them the calvarium may appear small (Plate 26) In *achondroplasia* the skull though of approximately normal size appears very large in contrast to the generally small stature In addition the bridge of the nose is greatly depressed and the nostrils tend to point directly forwards



Spa t c l pl g a



Pa k nson m



Ank lo ng
Sp n l t s



Old Rickets

Plate 24 —ATTITUDE



Hydrocephalus



Rickets



Osteitis Deformans



Acromegaly

Plate 25 —SKULL

In *osteitis deformans* (Paget s disease) in addition to the widening and bowing of the long bones the skull is often greatly enlarged particularly in its transverse diameter so that it appears to bulge above the ears (Plate 26)

2 THE JOINTS

These should be examined by inspection and palpation and by tests for their range of movement It is important to proceed in a routine manner—e g the jaw cervical spine shoulder girdle and upper limb thoracic and lumbar spine pelvis and lower limb so that inconspicuous but important joints like the temporo mandibular sterno clavicular and sacro iliac will not be overlooked and always to compare the corresponding joints on the two sides of the body

On inspection and palpation look for enlargement or irregularity of the joint for redness tenderness and heat indicating an acute arthritis for bony outgrowths such as Heberden s nodes on the fingers in some cases of osteo arthritis for gouty tophi and for atrophy of muscles in the region of the joint If the joint is enlarged determine whether the enlargement is due to effusion into the joint space when it normally has a characteristic shape and fluctuation can often be elicited to thickening of the periarticular tissues such as occur in the rheumatoid type of arthritis to enlargement of the ends of the bones such as occur in pulmonary osteo arthropathy or to complete disorganization of the joint with absence of pain sense such as occurs in neuropathic (Charcot s) joints If tenderness is present localize it as accurately as possible and determine particularly whether it arises in the joint or in neighbouring structures e g in the subacromial bursa rather than in the shoulder joint Feel the joint with one hand while it is moved passively with the other A grating or creaking sensation known as *crepitus* may be felt This often indicates osteo arthritis but not invariably so for crepitus is commonly felt in the shoulder joints of older persons whereas osteo arthritis of these joints is rare

In examining joints for the range of movement an estimate of the degree of limitation present based on previous experience or on comparison with the normal side may often be sufficient but for accurate description the actual range of movement should be measured with a protractor or goniometer In either case it is

important that the student should be able to describe the movements of the various joints correctly In carrying out the examination both active and passive movement should be tested where possible and the greatest possible gentleness must be exercised particularly in the case of painful joints Limitation of movement *in a joint may be due to pain muscle spasm contracture* inflammation or thickening of the capsules or periarticular structures effusion into the joint space bony or cartilaginous overgrowths bony ankylosis or to painful conditions quite unconnected with the joint

In describing the range of movement of joints the scheme shown in the following pages (modified by permission of the authors and publishers from Cave E F and Sumner M R *Journ Bone and Joint Surg* 1936 XVIII 455) will be found useful All motion should be measured in degrees from a neutral position or zero which must be defined whenever possible

Spine —Neutral position is normal upright position for patient, but cannot be further defined Test —

(1) Forward bending
(2) Extension
(3) Lateral bending
(4) Rotation with pelvis fixed comparing angle made between axis of shoulders and that of the pelvis

These movements cannot conveniently be measured but should be compared with the probable normal for the patient s age

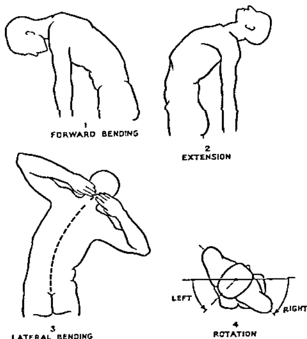


Fig 84

Neck —Neutral position is that with head erect and chin drawn in Test —

(1) Rotation right and left
(2) Flexion
(3) Extension
(4) Lateral bending

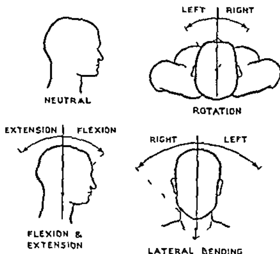
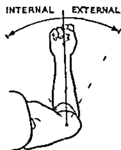


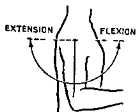
Fig 85

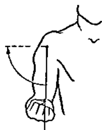
Shoulder —Neutral position is arm to side elbow flexed to 90 degrees with forearm pointing forwards Test —

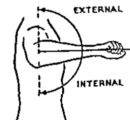
(1) Flexion
(2) Extension
(3) Abduction
(4) Rotation in abduction
(5) Rotation in neutral
(6) Elevation (this is shoulder girdle motion as compared with 1-5 which are humeroscapular motion)


NEUTRAL


INTERNAL
EXTERNAL
ROTATION IN NEUTRAL


EXTENSION
FLEXION
FLEXION & EXTENSION


ABDUCTION


EXTERNAL
INTERNAL
ROTATION IN ABDUCTION


ELEVATION

Fig 86

Elbow —Neutral position is with forearm in extension Test —

(1) Flexion
(2) Hyperextension
(3) Supination {from neutral which is mid point between supination and pronation with elbow fixed at side
(4) Pronation { in 90 degrees of flexion

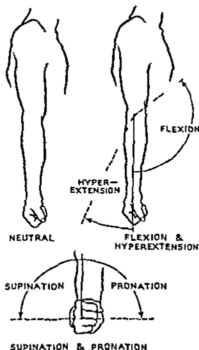


Fig 87

Wrist—Neutral position is with hand in line with forearm and palm down Test —

(1) Dorsiflexion (extension)
(2) Palmar flexion
(3) Ulnar deviation
(4) Radial deviation

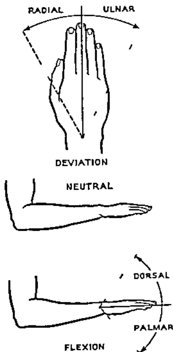


Fig 88

Fingers —Neutral position is with fingers in extension Test —

(1) Flexion at metacarpo phalangeal and inter phalangeal joints

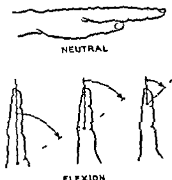


Fig 89

Thumb —Neutral position is with thumb alongside forefinger and extended Test —

(1) Abduction
(2) Flexion—measured as for the fingers
(3) Opposition

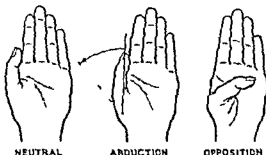


Fig 90

Hip —Neutral position is with hip in extension patella pointing forwards Test —

(1) Flexion measured with knee bent Opposite thigh must remain in neutral position
(2) Abduction measured from a line which forms an angle of 90 degrees with a line joining the anterior superior spines
(3) Adduction measured in the same manner
(4) Rotation in flexion
(5) Rotation in extension

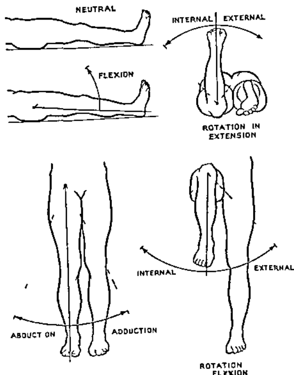


Fig 91

Knee —Neutral position is complete extension Test —

(1) Flexion

(2) Hyperextension

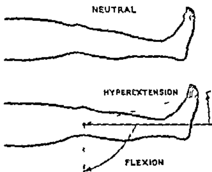
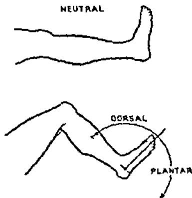


Fig 92

Ankle —Neutral position is with the outer border of foot at angle of 90 degrees with the leg and midway between inversion and eversion Test —

(1) Dorsiflexion with foot in inversion Test with knee in flexion and extension to exclude tight calf muscles

(2) Plantar flexion



Foot —Neutral position cannot be defined Test —

(1) Inversion and eversion at sub astragalar joints
(2) Forefoot adduction and abduction at mid tarsal joints with os calcis head in neutral position
(3) Flexion at metatarso phalangeal and inter phalangeal joints

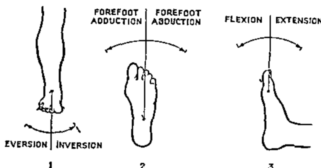


Fig 94

3 THE GAIT

The character of a patient s gait is often an important indication of the nature of the affection from which he is suffering It is especially important in cases of nervous disease

In studying the gait it is well to have the legs fully exposed The patient should therefore either be unclothed or wear a small triangle or bathing dress The feet should be bare The patient is told to walk away from the observer to turn round at a given point and then to come towards him again

In studying the gait the points to be noted are—(1) Can the patient walk at all? If he can—(2) Does he pursue a straight line or does he tend to deviate to one side or the other or to both alternately? To bring out this point ask him to walk along a straight line—e g a crack in the floor (3) Does he tend to fall and if so in what direction? The next point to be decided is whether the gait conforms to any of the well recognized abnormal types Before deciding this be quite sure that the peculiarity in the patient s gait

is not due to some surgical cause or to local disease of a joint—e g osteo arthritis of the hip A previous examination of the bones and joints will eliminate such sources of error

The three chief types of abnormal gait due to nervous affections are —

1 The spastic
2 The ataxic
3 The reeling

It is usually sufficient to state that the gait belongs to one or other of these types or to two or more combined The chief peculiarities of each variety are as follows —

1 The spastic may be described as a sticky gait The patient has difficulty in bending his knees and drags his feet along as if they were glued to the floor the toes scraping the ground at each step The foot is raised from the ground by tilting the pelvis and the leg is then swung forwards so that the foot tends to describe an arc

This gait is seen most characteristically in patients with pyramidal lesions The hemiplegic gait is essentially a spastic gait in which only one leg is affected

2 The gait in sensory ataxia may be described as stamping The patient raises his feet very suddenly often abnormally high and then jerks them forward bringing them to the ground again with a stamp and often heel first He may be fairly steady if he watches the ground as he uses his eyes in place of his sense of position but becomes severely ataxic when his eyes are closed This gait is best seen is cases of tabes dorsalis and other signs of loss of postural sensibility will be present

3 The gait of cerebellar inco-ordination may be described as a drunken or reeling gait and requires no further description Patients with this gait walk on a broad base the feet being planted widely apart The ataxia is equally severe whether the eyes are open or closed

This gait occurs in disease of the cerebellum or cerebellar tracts and other signs of motor ataxia will be present A similar reeling gait may be seen in patients under the influence of alcohol or narcotic drugs

Some rarer varieties of abnormal gait are —

The festinant gait This is met with in typical cases of paralysis agitans The patient is bent forwards and advances with rapid

short shuffling steps so that he looks as if he were trying to catch his centre of gravity and the arms do not swing In some cases if he is suddenly pulled backwards he begins to walk backwards and is unable to stop himself though he is leaning forwards all the time This peculiar phenomenon is spoken of as *retropulsion*

The **waddling** or oscillating gait is like the gait of a duck The body is usually tilted backwards there being a degree of lordosis present the feet are planted rather widely apart and the body sways more or less from side to side as each step is taken The heels and the toes tend to be brought down simultaneously The chief peculiarities of this gait are due to a difficulty in maintaining the centre of gravity of the body owing to weakness of the muscles of the back It is met with in pseudo hypertrophic muscular dystrophy and in congenital dislocation of the hip

The **high-stepping** or prancing gait is a device adopted by the patient to avoid tripping from his toes catching the ground It is therefore met with in cases where the toes tend to droop from weakness of the extensor muscles e g in peripheral neuritis affecting the anterior tibial nerve The name sufficiently describes its characters

CHAPTER XII

CLINICAL EXAMINATION OF CHILDREN

THE clinical examination of young children is a matter of difficulty to the inexperienced for it often has to be carried out in spite of the patient s strenuous opposition

The history of the patient and his illness must in the case of young children be ascertained from the mother or friends The best scheme of interrogation to employ will be found on p 11 Whilst the history is being elicited opportunity may be taken to cultivate the friendship of the child or at all events to get him accustomed to one s presence One then proceeds to an examination This requires gentleness and deliberation—combined with infinite patience and good temper If one is hurried or rough the child begins to cry at once and the subsequent examination is rendered much more difficult It is often not possible to be really systematic in one s examination of children Certain things must always be looked for but no definite order can be observed in looking for them One has to seize the opportunity of ascertaining a fact as it presents itself and a rigorous adherence to systems is often out of the question A number of points can be ascertained before the child is undressed One can study the facies of the child note his complexion the colour of his lips and whether or not the alæ nasi are acting One should also at this time count the respiration and pulse rate it is important to do this while the child is still undisturbed

The respirations can usually be counted by merely watching the movements of the child s abdomen which is much more affected by respiration in young children than the chest The normal rate of a new born child is 40 or so respirations per minute by the second year they have fallen to 30 or so at the fifth year they are about 25 and by the fifteenth they have sunk to 20 Much more important than the absolute number of respirations is the ratio of respiration to pulse Normally this should be as 1 3½ or 4

The pulse is best counted by allowing the mother to hold the child s hand in hers the fingers of the physician are then quietly

slipped over the mother s hand on to the child s wrist and the pulse counted If the child has begun to cry it is useless to take the pulse rate as it may be at least 20 beats above the normal rate The pulse rate at birth should be 130 by the second year it is 110 by the fifth 100 by the eighth 90 and by the twelfth 80 after this it gradually sinks to the normal adult rate During sleep the pulse rate always falls about 10–20 beats The examination of the pulse in infancy is of comparatively little clinical value Examination of the fontanelle is of much more use The vessel being extremely small the characters of the pulse wave can hardly be ascertained irregularity by itself is of comparatively little significance being very common even in healthy infants and being almost the rule in sleep A pulse that is continuously *slow* and irregular is however of great significance

The child should then be stripped and placed in a blanket on the knee of the mother or nurse examination should then proceed by the usual methods of inspection palpation auscultation and percussion In the clinical investigation of children the two former methods are of much the greatest assistance Begin by looking at and feeling the child all over Note the general state of development and nutrition the state of the skin whether dry and fevered or moist and the presence or absence of any rash or skin eruptions and whether or not the normal degree of elasticity is present The shape of the chest and the degree of prominence of the abdomen should be noted remembering that rather protuberant abdomen is to be regarded as normal in young children The hand should then be passed lightly over the head The state of the anterior fontanelle should first be investigated The fontanelle closes normally between the fifteenth month and the second year If it remains patent after the second year it is often a sign of disease—usually of rickets Too early closure of the fontanelle occurs in some forms of microcephaly and idiocy

The degree of tension of the fontanelle is of great importance In health it pulsates distinctly and is neither sunken or unduly elevated A depressed fontanelle is an important sign of exhaustion and of dehydration a tense fontanelle indicates increased intracranial pressure It must be borne in mind that the fontanelle is normally tense when the child is crying The systolic bruit heard over the fontanelle is of no clinical importance

The shape of the head and of its bones must be investigated

The development of 'bosses' on the frontal and parietal bones is a common occurrence in rickets One should seek evidence of craniotabes (in young babies) and of rheumatic nodules (in older children) The general shape of the head as a whole should always be noted it may be box shaped as in rickets or globular as in hydrocephalus it may be abnormally small or large or it may be asymmetrical

The examination of the limbs which should be carried out next is in children of extreme importance many of the commonest and most serious diseases of infancy affect the long bones more prominently than any other part of the body Look for thickening or tenderness along the shafts of the bones This may be due to scurvy to syphilitic or to suppurative periostitis or to tumours Examine carefully the epiphyses In rickets these become enlarged This is most easily seen where the ribs join their cartilages the thickening there forming a row of bead like prominences (*rickety rosary*) It is also easily seen at the wrists The frequency of inflammatory affections of the epiphyses should be remembered The presence or absence of *rheumatic nodules* should be noted These are little fibrous bodies varying in size from that of a large pin s head to a pea or even bigger They occur not in the perios teum but in the deep fascia where it covers superficial bones and also in the sheaths of tendons They are most common over the olecranon patella and occiput They are usually movable but not tender If found they are pathognomonic of rheumatism The **vertebral column** should always be examined for signs of tuberculous disease or curvature

The child s **temperature** should now be taken In young children the thermometer should be inserted into the rectum or placed on the groin or axilla in older children it may be placed in the mouth It should be remembered that the temperature in children is much more variable than in adults and that it often rises on very little provocation The rectal temperature is normally somewhat higher than the mouth or axillary temperature

One must now proceed to the examination of the **thorax and abdomen** The front of the chest and abdomen may be examined together and either after or before the posterior aspect of the chest The *order* adopted should be first inspection and palpation then auscultation and last of all percussion Percussion is *left to* the last owing to the fact that it frequently makes the child cry

In **palpation** be sure that the hand is quite warm this is even more important in examining a child than in the case of an adult It is also important to watch the child s face while this examination is being carried out, as wincing will provide evidence of tenderness and pain In **auscultation** it is important to warm the chest piece of the stethoscope if it is made of metal before applying it to the chest There is only one point to be observed in the **percussion** of a child and that is that the stroke should be *light* This is not merely in order to avoid frightening the little patient but also to escape the confusion that is apt to arise from the excessive resonance of the child s chest

The posterior aspect of the lungs should now be examined For this the child should not be laid on his face as this interferes with respiration and causes the abdominal viscera to push up the diaphragm but he should be held against the mother s breast with his head looking over her shoulder In this way the whole of the back of the chest can be gone over Enlarged glands should be sought for particular attention being paid to the neck which should be palpated from behind

Lastly comes the examination of the **mouth and throat** It is impossible to exaggerate the importance of systematically examining the mouth and throat in all cases of illness in children At the same time it is just this part of the clinical examination in which most opposition is likely to be met for that reason it is left to the last as it may be necessary to employ coercion in order to carry it out

Begin by looking at the **tongue** Sometimes the child will put out the tongue when asked In little babies gentle pressure on the chin will often cause the mouth to be opened when a view of the tongue can be obtained Or if a drop of milk or a little sugar is placed just outside the lip the child will often put out its tongue in order to lick it off In more refractory children it may be necessary to push the lower lip over the teeth and then to press the lip down against the lower incisors The child then opens the mouth in order to avoid biting the lip With very obstinate children it may be necessary to compress the nostrils until the mouth is opened to get breath

Once the child has been induced to open the mouth note the state of the **buccal mucous membrane** remembering the frequency of thrush stomatitis and ulcerations in children In cases

suspected measles *Koplik's spots* should be carefully looked for. They are irregularly stellate or round rose red spots with a bluish white speck in the centre of each. They are found on the inside of the lips and on the buccal mucous membrane especially opposite the upper molars. At first they are very sparse but later on become more numerous and the red parts may then coalesce into large areas dotted with the bluish white specks. They should always be looked for in strong sunlight if possible and never by artificial light. They are of considerable diagnostic importance for they may precede the appearance of the skin eruption by three or four days. The number and character of the teeth should be observed (*see also* p 33) and the finger should be run along the gum to feel for any teeth that may be about to come through.

One then proceeds to an examination of the throat. If possible the child should be held quietly by his mother sitting on her lap facing a good light. The observer should steady the child's head with one hand and gently insert a spatula with the other. Only if this technique is impossible should the child be wrapped in a towel to restrain the movements of its arms. The finger will often serve instead of a spatula and usually frightens the child less. Look for any enlargement of the tonsils for any redness of the mucous membrane and especially for the presence on it of any membranous patches.

The following is the general routine method employed in examining a child —

Facies and expression—general appearance (*if healthy or otherwise*)—nutrition, muscle tone, dentition and development—complexion (anæmia, cyanosis, jaundice, etc)—state of skin (dryness, *moisture*, eruptions, desquamation, pigmentation, œdema)—posture, demeanour, temper—pain on being handled

Shape of head and state of its ossification (fontanelle, craniotabes)—facial irritability—hair—eyes, nose and ears (formation of and if any discharge from)—shape of thorax, abdomen, back and limbs (especially the hands)—enlarged glands—evidence of rickets, syphilis and tuberculosis.

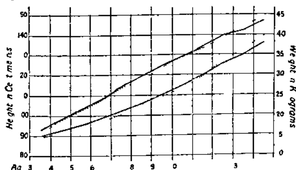
Character of voice, cry and cough—rate and character of respiration if noisy, dyspnœic or painful—movements of alæ nasi—rate and character of pulse—temperature

Palpation of abdomen (tenderness, resistance, fluid, size of liver and spleen, tumours, etc)

Certain special points are now considered briefly —

1 **General condition** —The we ght of children s of mportance in assess ng prognos s and treatment and a regular we ght record is of much value A healthy ch ld should we gh at b rth about 7 lb This should be doubled dur ng the s xth month and trebled in the first year By the sixth year t s aga n doubled so that a healthy child of six should we gh about 3 stones This is again doubled when the fourteenth year is reached (F g 95)

Measurement of the head s often of mportance Two measure ments are usually suffi ent a coronal measurement from one aud tory meatus to the other and a c cumferent al measurement



F g 95 Average he ght and we ght of English school-children

T f d d G gg

Th w pp line p h gh h w w in w gh Th in s o b y h d d in gir (ce tim 0 39 in. kil 2 b 3 o

(By permi f h B h A d J)

at the level of the root of the nose and external occ pital protuberance

2 **Alimentary system** —Note that the **liver** s normally large in children and often reaches at least 1 n below the costal marg n Enlargement of the **spleen** is frequent n infant le d seases It is best el c ted by palpat on the hand being passed across the child s abdomen from r ght to left By depress ng the finger tips oppos te the 11th nterspace the edge of the spleen f the organ is enlarged may be felt as t descends dur ng nspirat on When ind cated a rectal examinat on must not be omitted

Inspect on of the **stools** should be carr ed out The healt

infant on the breast or bottle only has generally two or three stools daily though there are wide variations These should be of the colour and consistence of beaten up eggs Any alterations in frequency colour or consistency or the presence of worms should be noted

3 **Circulatory system** —The apex beat of the child is normally rather higher than in the adult It is usually outside the mammary line up to the third year in the mammary line from the third to the tenth year after that age it gradually assumes the adult position Alterations in the general contour of the præcordia are much more frequent results of cardiac disease in children than in adults In **auscultation** the pulmonary second sound in a young child is normally rather louder than the aortic The pulmonary second sound is accentuated if it is permanently louder than the first The aortic second sound is accentuated if it is as loud as the pulmonary Hæmic bruits are rare in babies while congenital bruits are relatively frequent In this connection it is important to look for cyanosis and the presence of clubbing of fingers or toes

4 **The blood** —It is sometimes difficult to get a large enough drop of blood from the ear of a child In that case a piece of woollen thread should be twisted round the great toe—not too tightly—and the latter punctured with a triangular needle at the root of the nail The number of red cells is usually (after the second week of life) slightly lower than in the adult The leucocytes are more numerous in the child than in the adult 12 000 per c mm is about the average number throughout infancy The *lymphocytes* are both relatively and absolutely more abundant than in the adult amounting to about 45 per cent of the total leucocytes in the first three years In new born babies the percentage of hæmoglobin is often very high but throughout the rest of infancy it is lower than in the adult Nucleated red cells are present at birth and for some months afterwards The blood picture readily reverts to fœtal type in disease and the cells observed do not have the same ominous significance as they would if found in an adult

5 **Respiratory system** —A child uses the diaphragm much more than the intercostals in breathing Hence the movements are chiefly abdominal and there is little real chest expansion In

drawing of the lower interspaces on inspiration should always be looked for It occurs whenever there is obstruction to the entrance of air (e g diphtheria) but may also be present when there is collapse of the lower parts of the lungs and also in pneumonia One should be on the look out for any stridor and for the existence of a *short, grunting expiration and working of the alæ nasi* The latter is a frequent sign of severe respiratory disease In the adult the normal cycle of respiration is of course inspiration expiration, pause In the child this is often reversed so that one gets first a short expiration, succeeded by a longer inspiration and then by a pause This reversal is especially frequent in respiratory disease or embarrassment. The respiratory pauses are often very prolonged in the child so that one has to wait a long time before the next inspiration is heard The normal breath sound in the child is after the age of six months or so **puerile** in type **Vocal resonance** is often difficult to estimate and hence is an unreliable sign in childhood In babies the cry is a good producer of vocal resonance in older children one may ask them their name get them to count etc *It should be remembered as a general rule that if the breath* sounds are distinctly harsher on one side than the other then the harsh side is probably the normal Children s chests conduct sound very readily Hence abnormal sounds especially crepitations are apt to be heard on both sides although they are really being produced on one The great frequency of collapse of part of the lung should be borne in mind in diagnosing pulmonary disease in infancy In percussing the lungs in children one must *use a very light stroke* One should also take care only to percuss when the chest has been filled by an inspiration otherwise one may be led into thinking that there is dullness present The chest wall of a young child is so elastic that one can often obtain the cracked pot sound on heavy percussion even although the lung be perfectly healthy This is especially so if the child is crying

6 Urinary system —It is difficult to collect the total quantity of urine passed by a child per diem Sugar is only rarely present in the urine of children but protein is often found even in healthy babies

7 Nervous system —Motor paralysis is discovered by watching whether the child ever moves the suspected limb One cannot

estimate the paralysis as one does in adults by means of passive resistance Remember that inability to walk is not necessarily a sign of paralysis of the legs Note whether the legs are moved when the child is sitting or lying Thus a rickety child may not be able to walk but moves his legs freely if one tickles the soles A child with infantile paralysis of the legs cannot move the limbs in any circumstances

The **knee-jerks** in little children are best elicited by placing the child's foot on one's hand as a stirrup and then gently percussing the tendon The latter lies rather to the outer side in the child and is comparatively narrow so that one may easily miss it

The **superficial reflexes** are usually more brisk in healthy children than in adults The exact localization of sensory paralysis is extremely difficult in children but sensory lesions occur only rarely in infancy The plantar responses are normally extensor in type until the child begins to walk Kernig's sign (p 338) is quite unreliable in young children

In examining the eyes with the ophthalmoscope it may be necessary to hold open the lids but as far as possible avoid touching the child at all One must often be satisfied with mere fleeting glimpses of the disc

In testing light perception in little children it is best to hold a light in front of the eyes and see if they attempt to follow its movements One may also threaten the cornea by suddenly bringing the finger near it and observing whether the child winks before the eye is touched

In examining the **ears** one must remember the shortness of the auditory meatus in the child and the great obliquity of the drum membrane In most cases the drums of an infant or young child can be seen without the aid of an auriscope by gently pulling the pinna outwards and the tragus forwards

It is often difficult to gauge the **intellectual capacity** of a young child Early signs of mental deficiency are—inability to support the head which often rolls about helplessly causeless screaming inability to notice things and backwardness in grasping A normal infant will usually follow a light with its eyes at six weeks of age hold its head up at four months grasp at objects at six months sit up alone at nine months, crawl at about ten months walk at about twelve to fifteen months say words at about a

year and sentences at two years The age at which a child actually talks is very variable

In older children we can inquire as to progress at school etc or ask the patient questions get him to count multiply and so on The position of the child in the school is also a rough guide to the development of the intelligence For practical purposes it is true to say that a backward child would be normal for a younger age a defective one would be abnormal at any age

CHAPTER XIII

EXAMINATION OF PATHOLOGICAL FLUIDS

THE methods of examining fluids obtained from body cavities or from abnormal inclusions will now be discussed

An ordinary hypodermic needle may be used but should be of such calibre as to be capable of sucking up any fluid likely to be encountered Sterilize the needle by boiling for three minutes and clean patient s skin with some surgical spirit or iodine solution at the spot selected for puncture With a small hypodermic needle inject a few drops of 2 per cent procaine intradermally Through the wheal infiltrate the underlying tissues with procaine down to *the pleura the peritoneum or the pericardium according to the* particular puncture that is being attempted Then wait two or three minutes so that the subsequent exploration is not unnecessarily painful Hold the exploring needle short with the forefinger resting on it near the point Introduce it rapidly and steadily but without any stab When the needle is fully entered withdraw the piston of the syringe If no fluid is obtained draw the needle slowly outwards whilst a negative pressure is maintained in the syringe It may be found that fluid is obtained nearer the surface

1 WHERE TO PUNCTURE

In the case of the pleural cavity the site depends on the position of the fluid If fluid is free the 5th or 6th space in the midaxillary line or the 8th space just below the tip of the scapula are the sites of election If the fluid is encysted the puncture may be made over the area of maximum dullness In the case of encysted effusions however the exact position of the fluid should if possible be previously determined by means of a postero anterior and lateral X ray of the chest

Puncture of the peritoneal cavity may be performed either in the middle line through the linea alba or laterally about a point on a line with but rather above the anterior superior spine The

former position ensures that no large blood vessel will be injured but by lateral puncture there is more certainty of entering fluid especially if the patient is turned over somewhat on to the side of operations Before puncturing be sure the bladder is empty and never insert a needle at any point unless it yields a dull note on moderately heavy percussion

The pericardial sac may be entered by the epigastric or lateral thoracic route In the former the needle is entered just below and to the left of the xiphisternum and is directed upwards and slightly to the left at an angle of 30 to 40 degrees to the skin In the latter the needle is entered in the 5th left intercostal space just internal to the lateral border of the cardiac dullness and directed back wards and inwards towards the spine

In the exploration of cysts etc one must be guided by local circumstances the rule being to select for puncture that part of the tumour which is nearest the surface and where one is not likely to injure important structures

EXPLORATION OF LIVER FOR LIVER ABSCESS

Liver abscesses due to the *Entamœba histolytica* are nearly always found in the right lobe of the liver This condition should be suspected whenever a patient who has had amœbic dysentery or who has lived in the tropics or sub tropics shows fever tenderness in the region of the liver and signs of enlargement of the liver either upwards or downwards or both Sometimes there is an area of maximum tenderness in a lower right intercostal space and if this is the case the area should be carefully marked These signs are sometimes due to amœbic hepatitis without abscess formation The patient should therefore first be treated with emetine hydrochloride If the signs persist or become more marked under this treatment exploration of the liver for diagnostic and thera peutic purposes should not be too long delayed The method is as follows The main danger is that the needle will enter the inferior vena cava Puncture of the intrahepatic branches of the portal vein does no harm It has been shown that in a man with a chest of 32 in circum ference the inferior vena cava is nowhere less than 4½ in from the surface A needle of wid enough bore to admit thick pus (a stout old fashioned lumbar puncture needle is suitable) is selected and a piece of adhesive tape is wound around it 3½ in from its point After anæsthetization of the skin and subcutaneous tissues the needle is entered either at the site of maximum tenderness already noted or in the 8th 9th or 10th space in the right axilla and passed laterally i e towards the patient s opposite side and slightly upwards i e towards the patient s head to a maximum

depth of 3½ in By this means the whole of the right lobe of the liver can be explored The pus in a liver abscess may be thick Thus as already mentioned a stout needle must be used and fairly strong suction maintained with the syringe

If pus is encountered it is usual to remove as much as possible with the aid of a two-way syringe As the pus is removed it may be replaced by air until either the patient complains of pain in the liver or shoulder or until half of the volume of pus removed has been replaced by air If the patient is then X rayed in several positions the exact site and size of the abscess can be determined and the effect of treatment on its size can be followed

2 EXAMINATION OF THE FLUID

Note first its physical characters The chief of these are the colour and the appearance of the deposit (if any)

As regards the colour of the fluid one of the most important points to note is whether it is blood stained or not A small amount of blood is apt to get into the fluid in the process of exploring For this reason samples of the fluid should be taken into several test tubes If the amount of blood diminishes in the later tubes its presence is due to trauma at the time of puncture Observe also whether the fluid is transparent opaque or opalescent Many pathological fluids clot after standing for some time The clot consists of fibrin

Opacity is usually due to the presence of pus opalescence to fatty particles or large numbers of micro organisms

Opacity or opalescence due to fat may be removed by adding to the fluid some caustic potash solution then shaking up with ether The fat is dissolved out and if the ether is sprinkled on to blotting paper leaves a stain Fluid which is opaque from the presence of much fat is usually spoken of as chylous It is found in obstruction to the thoracic duct It may be simulated very closely by a pseudo-chylous fluid in which the milkiness is due to a lecithin globulin complex which results from the degeneration of cells shed into the effusion

3 MICROSCOPICAL EXAMINATION OF THE SEDIMENT

Some of the deposit is taken up with a pipette and a drop placed on a slide covered and examined For cytological ex

amination it is best to centrifuge some of the fluid and make films from the deposit. The films may be stained with carbol thionin or *Leishman's stain*

There may be recognized under the microscope (1) elements derived from the blood—altered red and white corpuscles The recognition of altered white corpuscles or pus cells is facilitated by mixing with a drop of the deposit a small quantity of a 1 per cent solution of acetic acid to which a little methyl green has been added The nuclei are then easily recognized In acute inflammatory exudates polymorphonuclear cells predominate in tuberculosis and syphilitic infections lymphocytes are in excess (2) Endothelial cells —These are derived from the lining of the cavity and are the only cells present in passive transudates Cancer-cells may be present in malignant cases but it is difficult to distinguish them with certainty from ordinary cells In fluid derived from hydatid cysts scolices and *hooklets* may be found (3) **Crystals** of cholesterol or of fatty acids and fragments of muscular tissue are sometimes seen

Though cancer cells can rarely be recognized for certain in stained films fragments of growth can sometimes be recognized by using histological methods For this purpose the fluid is centrifuged in a flat bottomed tube The deposit is then fixed in formal saline passed through alcohols cleared in xylol and embedded in paraffin Sections are cut and stained by the usual histological methods and fragments of growth may then be recognized

The **bacteriological examination** of pathological fluids is described in Chap XIV

4 INFLAMMATORY AND PASSIVE EFFUSIONS

Inflammatory effusions such as occur in tuberculosis are often spoken of as exudates and passive effusions such as are found in heart failure and nephritis with œdema as transudates They present the same general appearances being clear fluids of a yellowish green colour and containing much albumin and globulin It is very difficult to tell a passive from an inflammatory fluid by chemical examination one must rely on cytology The amount of proteins in an effusion depends much more upon site than upon cause Pleural fluids contain the highest percentage of protein

peritoneal fluids rather less and subcutaneous fluids very little From a diagnostic point of view all that can be said is that a fluid with a specific gravity of more than 1018 which contains more than 4 per cent of protein is almost certainly inflammatory while one with a specific gravity of less than 1015 and a protein percentage of less than 2 5 is certainly passive Between these limits one must be in doubt

5 CEREBRO SPINAL FLUID

The fluid is obtained by **lumbar puncture** which is performed as follows —

Draw a line with a swab dipped in alcoholic solution of iodine vertically down the vertebral spines and another horizontally at the level of the highest points of the iliac crests The lines intersect at the space between the 3rd and 4th lumbar spines or sometimes at the tip of the 4th lumbar The puncture may be made through either the 3rd or 4th interspace The patient should be lying on his side on a firm couch with the knees and chin as nearly approximated as possible His back should be right at the edge of the couch and it is important that its transverse axis i e a line passing through the posterior superior iliac spines should be quite vertical Local anæsthesia may be produced by injecting 2 per cent sterile procaine first raising a bleb under the skin and when this is insensitive thrusting the needle in towards the centre of the intervertebral space injecting the solution as one does so A special platinum iridium or nickel needle about 8 cm in length should be employed (steel is too brittle) it should be of fine calibre and provided with a bevelled end and a stylet It may be mounted for convenience (but not for suction) on an all glass syringe of 10 ml capacity It may be sterilized by boiling in distilled water but sterilization in an autoclave is safer

Push the needle firmly through the skin in the middle line or just to one side of it and press it forwards and slightly upwards the bevel pointing towards the side on which the patient is lying When the needle is felt to enter the spinal cavity the stylet is withdrawn and the fluid which escapes collected in sterilized test tubes The puncture is sealed with collodion and the patient should lie flat for 24 hours afterwards The fluid should be collected in three test tubes If any blood is present a marked difference in the

amount in the first and third tubes indicates that the blood is due to trauma from the puncture

It is an advantage to have a manometer connected with the needle so that the pressure of the fluid can be measured at the time of puncture If this is done the patient s head must be on the same level as the sacrum and he must be *breathing quietly* and with his muscles relaxed The normal pressure is from 60 to 150 mm of fluid

Queckenstedt s test is used to detect a block to the circulation of fluid in the spinal cord With the needle and manometer in position and the patient breathing quietly as described an assistant compresses both jugular veins This causes a sudden increase in intracranial pressure which is immediately seen in the manometer as a sudden rise of cerebro spinal fluid pressure followed by an equally rapid fall when the pressure on the jugular is released A similar sudden rise and fall is seen if the patient is asked to cough *With slight degrees of blocking there may be a rise of pressure* in the manometer followed by a very slow fall when the pressure on the jugular is released and with more severe blocking no rise of pressure will be seen when the jugulars are compressed

Much the commonest cause of a dry tap the failure to obtain fluid is an incorrectly performed puncture and this is usually due to the patient not being in the correct position so that the needle is *not introduced at right angles to the transverse axis of the back* and misses the spinal canal Occasionally however a dry tap is due to a complete blockage of the spinal canal and under these circumstances it may be necessary to resort to cisternal puncture to obtain a specimen of cerebro spinal fluid This operation presents no great technical difficulty but should only be under taken when lumbar puncture attempted by a competent operator has been unsuccessful and when there is no reason to suspect a tumour or abscess of the posterior fossa or a great increase of intracranial pressure The patient s head is shaved for a few inches at the back He is seated and his head is held well flexed *between the hands of an assistant* The operator then finds the highest palpable spinous process which is that of the second cervical and injects some 2 per cent procaine into the skin half an inch above this process *A lumbar puncture needle is introduced* through this point and passed forwards in a plane which passes through the point of introduction the external auditory meatus

and the nasion At a point about 3 cm from the surface the resistance of the posterior occipito atlantal ligament is felt The needle is then introduced a little farther and should enter the cisterna magna The medulla lies at a depth of about 3 cm in front of the posterior occipito atlantal ligament A piece of adhesive tape wound around the needle 4 5 or 5 0 cm from its point will prevent any danger of the medulla being damaged

Neither lumbar nor cisternal puncture should be performed in patients with papilloedema nor in patients otherwise under suspicion of having a cerebral tumour until a neuro surgeon has been consulted Lumbar puncture should also be avoided in patients with disseminated sclerosis if the diagnosis can be made without it

6 EXAMINATION OF THE FLUID

1 **Physical characters** —Normal cerebro spinal fluid is clear and colourless like distilled water with a specific gravity of 1006 Any yellowness of tint is pathological and is due either to old hæmorrhage obstructive jaundice or excess of protein In *Froin s syndrome* a pronounced yellow colour (xanthochromia) is associated with great excess of protein and massive coagulation of the fluid Formation of a clot in a colourless fluid indicates meningitis usually tuberculous or high protein from other cause e g tumour or polyneuritis

Turbidity of the fluid may be due to pus or to red blood corpuscles If it does not clear on standing it is due to micro organisms

The presence of blood may be due to injury to a vessel by the needle or to subarachnoid hæmorrhage In the latter case the blood is more uniformly mixed with the fluid and the supernatant fluid after the corpuscles have settled is yellow

2 **Cytology** —If the fluid is turbid 5 ml should be centrifugalized and films made from the deposit and stained with Leishman s stain Examination of the film will give a rough idea of the number and character of the cells present More than five cells in a field may be regarded as pathological

To carry out a cell-count, take a capillary pipette and make a mark on it with a grease pencil about 3 cm from its distal end Using this marked off portion as a unit of volume take 4 volumes

of cerebro spinal fluid and one volume of any simple stain (Loeffler s methylene blue or carbol thionin) Mix well in a watch glass or clean test tube and place a suitably sized drop in a Thoma Zeiss counting-chamber

Adjust the draw tube of the microscope so that the diameter of the field with the $\frac{1}{6}$-in objective is 8 small squares Count the cells in 100 fields refilling the chamber when necessary Then the number of cells in 100 fields = the number in 1 c mm

For the area of one field 50 small squares (approximately)

50 small squares $\frac{50}{4\,000}$ c mm = $\frac{1}{80}$ c mm but the fluid is diluted to four fifths strength

$$100 \text{ fields} = \frac{100}{80} \times \frac{4}{5} \text{ c mm} \quad 1 \text{ c mm}$$

A special counting-chamber (e g the Neubauer) may also be used

It should be noted tha a cell-count must be done immediately the fluid has been collected Counts done some hours later give inaccurate results owing to the fact that pus cells stick together and to the sides of the tube while endothelial cells break up in a short time If any clot has formed an accurate cell-count cannot be obtained

An excess of cells (pleocytosis) is described as being of the polymorphonuclear type if these cells are above 75 per cent of the total and of the lymphocyte type if more than 90 per cent are lymphocytes A mixed type also occurs in which the poly morphs amount to from 15 to 70 per cent of the total The coccal forms of meningitis are associated with the polymorphonuclear type virus meningitis and syphilis with the lymphocytic type and tuberculous meningitis and poliomyelitis with either a lymphocytic or a mixed type

3 **Chemical examination** —(*a*) *Proteins* Normal cerebro spinal fluid contains only a trace of albumin and hardly any globulin the total protein being not more than 40 mg per 100 ml

The protein content can be roughly estimated by placing 2 ml of fluid in a test tube and carefully running in an equal quantity of absolute alcohol down the side In normal fluid the line of junction is just visible if protein is present in excess there is a

turbid ring The total protein is estimated more accurately by precipitation and comparison with standards

In certain pathological conditions the globulin fractions in the cerebro spinal fluid are altered The Lange test takes advantage of this fact varying dilutions of cerebro spinal fluid are mixed in ten tubes with a colloidal gold su pension of constant strength The degree of precipitation which results is expressed by arbitrary figures 0–5 0 representing no change and 5 complete precipitation

The Wassermann serological reaction is often performed on the cerebro spinal fluid

(*b*) *Glucose* Normal cerebro spinal fluid contains from 50 to 75 mg glucose per 100 ml which is less than the amount of sugar in the blood

If 1 ml of fluid is boiled with 0 25 ml of Fehling s solution the latter should be almost decolorized if much blue is left it is an indication of reduced sugar content Accurate estimation of the sugar is carried out in the same way as in blood (p 171) The amount of sugar is diminished in acute purulent meningitis

(*c*) *Chlorides* Normal fluid contains from 0 72 to 0 75 per cent of sodium chloride Amounts below this are met with in cases of meningitis and above it in renal failure The low chlorides in cases of meningitis (classically tuberculous) are more likely a measure of general chloride deficiency due to dehydration than any specific local effect of the organism The chlorides are esti mated by adding 2 ml of fluid to 20 ml of distilled water and titrating with standard silver solution chromate of potash being used as indicator

For a table showing the typical changes in the cerebro spinal fluid in various diseases *see* the opposite page

TABLE S O VING T E TYP CAL C ANGES IN THE CEREBRO-SP NAL FLUID IN VAR OUS DISFASES

—	N m	M ng M go-	 T b	D m n d su s	G P I	Tab s	M nse yp l	A po y s
Phy h ra	C c g m	G g b b d y co gu m	C w h ob b g m	C o	C s m m fl g m	Cl	C o a b d m m s e g m	U ua y c a som me c bw b coag um
C m ph Lymphoc	0 {C 0 3{ c m	00 000 5 50	0 00 00 300	0 5 00	0 5 5 00	0 5 00	0 50 0 500	0 o 00 o h gh d fly p ym ph f Lymp ocy
To p n	0 35	0 00	50 00	30 60 y gh	40 00	30 60	0 00	30 o 60 y d ys g 00- 00 nd m ng h gh 8 o 0 k
G b — N e-Ap G uc Cl nd R	0 50 75 7 0 o 750	+ o + 0 15 600 o 680	+ + 5 50 480 650	N rm	45 60 N m +	45 o 0 N m + 8 0	± + N m	0 + m y be 00 N m
ture	S e	M go- occ	T B fl m me d m y be d y pec m h d	S	S	S	S o	S
		000 443 0		†	555543 00	0 3 0000	0 23 0000	

† A paretic type f c s f nd i 50 p c n f c es

CHAPTER XIV

SOME BACTERIOLOGICAL AND OTHER LABORATORY INVESTIGATIONS

THE methods of collecting material for bacteriological investigation the value of such investigations and some other laboratory techniques are now briefly discussed This in no way replaces textbooks of bacteriology which should be consulted for further details of bacteriological methods Many of the examinations particularly those involving cultivation of material on special media serological technique and injection of laboratory animals will be carried out in the bacteriological laboratory In such instances it is essential that the specimen sent to the laboratory should be labelled with the patient s name age and sex The duration of the illness the tentative diagnosis and a few notes on the patient s clinical conditions should be supplied In this way the examination of the specimen in the laboratory is greatly facilitated and the bacteriologist will be in a better position to indicate the possible significance of his findings The result of bacteriological investigations must finally be assessed in relation to the clinical condition by the physician in charge of the patient but it will often be helpful to have the opinion of the bacteriologist in the interpretation of the laboratory findings

The bacteriological laboratory may be of assistance by finding either an infecting organism or evidence of a specific infection in various serum and other reactions

1 COLLECTION OF SPECIMENS

All specimens should be taken with such precautions as will reduce to a minimum the chance of contamination from external sources but admixture with antiseptics must be avoided Specimens should be received directly into sterile vessels which are at once closed with suitable stoppers In cases where cultural methods or animal inoculations are to be employed the examinations

should be carried out as soon as possible after withdrawal of the material from the body as many pathogenic organisms soon die or if present in mixtures are overgrown Where fluid specimens are to be sent by post cotton wool plugs are obviously useless and should be replaced by sterile rubber stoppers the containers should be suitably packed in absorbent material and the package labelled pathological specimen with care

2 BLOOD

A specimen of blood may be taken for culture or to have the serum examined for specific antibody

Blood culture is indicated when bacteriæmia is suspected. In this condition bacteria are present in the circulating blood reaching it from a poorly localized focus of infection Bacteriæmia may be suspected in local pyogenic infections where the temperature and pulse rate fail to settle down Typhoid or paratyphoid bacilli are always present in the blood in the early stages of enteric fever and the infecting organisms may be isolated by blood culture in cases of undulant fever infective endocarditis and lobar pneumonia In cases of pyrexia of unknown origin the isolation of an organism may establish the diagnosis and samples of blood should if possible be taken before any antibiotic is administered

3 METHOD OF OBTAINING BLOOD FOR CULTIVATION

The blood should be withdrawn from a vein by means of a 10 ml syringe of Record or all glass type The separate parts of the syringe should be sterilized by boiling in water for ten minutes before use When cool the parts should be put together by means of sterile forceps and care should be taken that no water remains in the syringe as this would tend to produce laking of the blood

A piece of rubber tubing is used as a tourniquet and applied round the upper arm over the middle of the biceps so as to impede the venous but not the arterial flow The skin at the bend of the elbow is painted with iodine in spirit The skin is rendered tense by the operator s left hand the syringe with needle attached is held in the right hand and almost parallel with the patient s arm

then the needle with the bevel upwards is inserted into a prominent vein The median basilic is usually selected and the needle is pointed in the direction of the blood flow Then 5 to 10 ml of blood is drawn up into the syringe and the tourniquet is removed before the needle is withdrawn as otherwise a hæmatoma tends to form The blood is at once placed into suitable tubes or flasks of medium It may be desirable to ascertain the number of living bacteria in the blood and for this purpose the specimen should be mixed with an equal volume of a sterile solution of 0 5 per cent sodium citrate in 0 6 per cent sodium chloride Coagulation is thus prevented and known quantities of blood can then be incorporated in a suitable medium

Success in the demonstration of bacteria which are present in the blood stream depends on the number present the amount of blood taken for culture the use of suitable media and the conditions of cultivation For most purposes the volume of medium used should be at least ten times the amount of blood added e g 10 ml of blood to 100 ml of culture medium The medium may with advantage contain trypsin or some other substance to prevent the clotting of the added blood and to destroy the antibacterial properties Where bacteria are few in number several days may be required before they are detected in culture

It is usually advisable to put up anaerobic cultures as well as aerobic ones For slope or plate cultures the simplest device is the jar of McIntosh and Fildes in which the oxygen is removed by reacting with a stream of hydrogen with a catalyst For fluid media the easiest method is to boil the medium to drive off the air and while still hot to pour a ½-in layer of sterile petroleum jelly on its surface The medium can be inoculated through the petroleum jelly at the side of the tube with a Pasteur pipette care being taken to introduce no air when expelling the pipette contents

In suspected cases of Weil s disease the specimen of blood should be added to citrate to prevent clotting The causal organism (*Leptospira icterohæmorrhagiæ*) will not grow in ordinary culture media but may be detected by special cultural methods or by animal inoculation

Bacteriæmia is usually present in infections with *Bact typhosum Bact paratyphosum A and B Brucella abortus Brucella melitensis* and the leptospira of Weil s disease and may be a complication of infections due to streptococci staphylococci pneumococci and

meningococci Other organisms such as *B coli* the gonococcus *Cl welchii* and the anthrax bacillus are occasionally found in the blood stream in unusually severe infections caused by them

The identification of an organism isolated by blood culture may serve to determine the nature of an infective condition and also as a guide for specific treatment Once an organism is isolated by blood *culture or other methods and specific treatment is con*templated the sensitivity of the organism to antibiotics should if possible be determined before treatment is begun though in some instances it may be justifiable to begin treatment with simple antibiotics (penicillin or sulphonamides) without bacteriological control

4 CEREBRO SPINAL FLUID

Examination of the cerebro spinal fluid by bacteriological methods should be made in any patient in whom meningitis is suspected

The technique of lumbar puncture has already been described In cases of meningitis the number of cells is always increased and except in meningitis of tuberculous or syphilitic origin and in the rare cases of meningitis due to virus infection the fluid is usually turbid and may be frankly purulent Films should be made from the fluid after centrifugation if necessary and stained by Gram s and by Ziehl Neel en s methods Bacteria may be recognized in films and the results of this examination will indicate what cultural methods are to be adopted for the isolation and identification of the infecting organism

In cases of tuberculous meningitis the bacilli are usually scanty and may be found in films of the centrifuge deposit only after prolonged search Better results may be obtained if the fluid is allowed to stand at room temperature for some hours when a delicate cobweb clot of characteristic appearance frequently forms A film made from the clot and stained by Ziehl Neelsen s method will usually show acid fast bacilli if careful search be made The organism requires several weeks before visible growth appears on suitable media and as the test by animal inoculation also takes three to six weeks these two methods are seldom of practical clinical value in cases of tuberculous meningitis

Meningitis may also be due to the meningococcus pneumococcus streptococcus staphylococcus *Hæmophilus influenzæ* and

less commonly to other organisms In these instances cultural methods should always be used to confirm and supplement the results obtained by microscopic examination of stained films of the fluid

5 SPUTUM

Bacteriological examination of the sputum is indicated in any inflammatory condition involving the trachea bronchi or lungs

The specimen is best collected first thing in the morning The mouth should be washed out with warm water so as to avoid as far as possible oral contamination and excessive mixture with saliva The specimen should be collected in sterile wide mouthed metal or glass containers

The bacteriological examination of sputum may be considered under two headings (i) for the tubercle bacillus and (ii) for other bacteria

If the tub rcle bacillus alone is to be sought for a specimen may be satisfactory even if it is two or three days old When however it is desired to obtain a true picture of the bacterial flora the specimen should reach the laboratory with as little delay as possible

1 Examination for tubercle bacillus —The tubercle bacillus is not readily culturable by ordinary methods but on the other hand it has characteristic staining qualities and is therefore sought for in film preparations

A mucopurulent portion may be selected and spread fairly thickly on a microscope slide Only new slides should be used More satisfactory results are obtained if the sample of sputum be mixed with 5 volumes of 1 in 20 carbolic acid in a stoppered container which is then thoroughly shaken The specimen is then allowed to settle overnight and the supernatant fluid is discarded The remainder is spun in the centrifuge for 5-10 minutes and films made from the deposit The film should be allowed to dry fixed by heat and stained by Ziehl Neelsen s method The tubercle bacillus being acid fast retains the fuchsin and shows up red against the blue background (*see* Plate 23 facing p 287)

In young children and infants who habitually swallow their sputum stomach washings may be examined

A single negative examination for tubercle bacilli should not carry great weight and the examination should be repeated several times in any case of suspected respiratory tuberculosis

If present in very small numbers tubercle bacilli may be demonstrated by animal inoculation This examination however entails a delay of 3-6 weeks before a final report can be made

2 **Examination for other bacteria** —For identification of other bacteria films of the sputum should be stained by Gram s method and cultures prepared on suitable media

In the bacteriological examination of a child suspected of suffering from whooping cough the coughplate method gives the best results A Petri dish containing a special potato extract blood agar is held in front of the patient s mouth during a paroxysm and is placed in an incubator at 37 C immediately afterwards The examination of the plate within the next few days may reveal the presence of *H pertussis* A better method of isolating this organism is to take a post nasal swab and plate it on Bordet Gengou medium that has been covered with penicillin solution

Sputum from normal individual may contain various bacteria such as streptococci pneumococci diphtheroid bacilli *Micrococcus catarrhalis* and *H influenzæ* Except in cases of respiratory tuberculosis acid fast bacilli are very rarely found in the sputum so that the findings of such organisms in a suspected case of pulmonary tuberculosis is of great diagnostic importance In attempting to assess the significance of other bacteria found in the sputum of patients suffering from respiratory tract infections one may be guided by the presence of pus cells and the relative numbers of various bacteria present

6 THE THROAT AND NASO PHARYNX

Bacteriological examination of the throat and naso pharynx may be desirable (*a*) in inflammatory conditions for identification of the infecting organisms and (*b*) in healthy individuals to determine the presence of pathogenic bacteria for example in searching for carriers of diphtheria bacilli hæmolytic streptococci or meningococci

Specimens should be obtained by rubbing the surface with a swab of cotton wool wrapped around the end of a strong wire

The swab is kept in a narrow test tube of stout glass and the other end of the wire may be fixed in the cork stopper The whole apparatus is sterilized by dry heat and kept ready for use In taking specimens it is important that no antiseptic should have been used (e g as a gargle) for some hours before Where inflammatory exudate is present the swab should be rubbed firmly over the affected area and passed under the edge of the membrane in suspected cases of diphtheria

The swab used for the naso pharynx is longer than that used for the throat and the wire is bent near the extremity A tongue depressor is used the wire is passed into the mouth and up behind the soft palate and then brought into contact with the posterior pharyngeal wall Care must be taken not to touch any part of the mucous membrane of the mouth

The method of examination of the material obtained varies according to the nature of the infection In suspected cases of diphtheria the swab is used to inoculate a slope of Loeffler s serum and if desired other special media containing tellurite The inoculated media are kept at 37 C and examined for the presence of diphtheria bacilli in suitably stained films after 6 to 18 hours The bacteriological findings must be evaluated in conjunction with the clinical condition and the final verdict must rest with the clinician In a small percentage of cas s of diphtheria the bacteriological report may be negative *when the clinical pictu e is that of a diphtheritic infection it is wiser not to wai for the bacteriological report before administering antitoxin*

In patients presenting the features of acute tonsillitis a Gram stained film of the material on the swab may indicate the nature of the infecting organism and confirmation should be sought by cultural methods using blood agar and Loeffler s serum

Vincent s angina may produce superficial ulceration of the throat and gums and may simulate diphtheria when the lesion is confined to the region of the tonsils Identification of the causal organisms should be made by the examination of stained films of the exudate The organisms associated with the condition a large Gram negative bacillus and a spirochæte do not grow on ordinary culture media For their demonstration, Gram s stain may be used but polychrome methylene blue or Leishman s stain give better results

The identification of meningococci in swabs from the naso-

pharynx involves culture on suitable media such as serum or blood agar and the isolation and identification of the Gram negative cocci by suitable tests These organisms tend to die out rapidly in material kept at room temperature so that it is essential that naso-pharyngeal swabs should be received and examined in the laboratory as soon as possible after they have been taken

For the detection of carriers the same general methods are used It should be noted however that for the identification of diphtheria bacilli in such instances microscopical examination of films from cultures must be confirmed by virulence tests in the guinea pig before organisms which resemble diphtheria bacilli can be reported as such The carrying out of a virulence test involves a delay of several days before a final report can be received

A variety of bacteria may be present in the normal throat and naso pharynx including diphtheroid bacilli pneumococci staphylococci streptococci influenza bacilli and Gram negative cocci Apart from the infections due to diphtheria bacilli and the organisms of Vincent s angina acute inflammation of the throat and tonsils is most commonly due to hæmolytic streptococci although *Staphylococcus aureus* pneumococcus and occasionally actinomyces and *Monilia albicans* may be responsible

7 NOSE AND NASAL SINUSES

Examination of material from the nose may be carried out as in the case of the throat to identify the organisms responsible for inflammatory conditions or to detect carriers of pathogenic bacteria among apparently healthy individuals

Material from the nose may be obtained by means of a throat swab In cases of sinus infection, where there is free discharge a specimen may be blown into a wide mouthed bottle or if the discharge is not free the sinus may be washed out with sterile saline and the washings sent for examination The identification of diphtheria bacilli in material obtained from the nose cannot be made by microscopic examination of films from cultures alone and must be confirmed where necessary by a virulence test For the identification of other bacteria in a nasal discharge films of the material should be stained by Gram s method and the results of the examination of such films should be supplemented by culture on appropriate media

In swabs taken from the healthy nose diphtheroid bacilli staphylococci pneumococci and Gram negative cocci may be present Inflammation of the nose and accessory sinuses is most frequently due to the pneumococcus hæmolytic streptococcus and *Staphylococcus aureus*

8 FÆCES

In any intestinal infection bacteriological investigation of the fæces may reveal the causal organism In suspected cases of enteric fever and dysentery this examination should always be made and is the best method for the detection of typhoid or dysentery carriers

A loose motion should be obtained and in the case of suspected carriers it may be necessary to administer a purgative Of a loose motion 1 ml is sufficient and any abnormal portion e g containing mucus or pus should be selected The usual sterilized throat swab soaked in fæces and replaced in its test tube is satisfactory when there is no delay in making the examination In other cases the specimen should be placed in a wide mouthed vessel by means of a small metal spoon fitted into the stopper In suspected cases of bacillary dysentery a satisfactory specimen may be obtained by means of a rectal swab from material taken from the mucous membrane of the rectum in this way dysentery bacilli may be recovered more readily than from a sample of stool

Where the making of cultures for organisms of the enteric and dysentery groups must be delayed for more than a few hours after the specimen has been taken it is advisable to add to one volume of the fæces two volumes of 30 per cent neutral glycerol in 0 6 per cent sodium chloride solution and to make a thorough mixture The presence of the glycerol prevents the suppression of the specific organisms by *B coli* which would otherwise occur

In the laboratory films of the specimen are stained by Ziehl Neelsen s method if the presence of tubercle bacilli is suspected When the examination of such films shows the presence of acid fast bacilli this result must be interpreted with caution for acid fast organisms other than the tubercle bacilli are sometimes found in the fæces The identification of tubercle bacilli in fæces will finally depend on the inoculation of animals or suitable media with a portion of the specimen treated by one of the methods devised to destroy other bacteria without killing tubercle bacilli

For the detection of bacilli of the enteric and dysentery groups a suspension of the fæces should be sown in a selective liquid medium or on a solid medium such as MacConkey s lactose bile salt agar The identification of these bacteria depends on their isolation in pure culture and the results of fermentation and agglutination tests Isolation of the infecting organism in cases of enteric fever or dysentery is not always successful The results will vary according to the relative number of these bacteria in the fæces the interval elapsing after the specimen has been obtained and before cultures have been set up the cultural methods used, and other factors In dysentery for example the specific bacteria may be difficult to isolate after the first few days of an attack especially if delay between the collection and cultural examination of the specimen occurs In such cases further specimens should be examined and after the first week of the disease indirect evidence of the nature of the infection may be obtained by examination of the serum for specific antibodies (*see* below) In the detection of carriers it may be noted that typhoid or dysentery bacilli may appear in the fæce only at irregular intervals and in small numbers It may therefore be necessary to have many specimens examined at weekly intervals before a carrier is detected

The fæces of normal individuals contain enormous numbers of various types of bacteria including lactose fermenting and late lactose fermenting coliform bacilli non hæmolytic streptococci, Gram-positive bacilli of the acidophilus type and various aerobic and anaerobic spore bearing bacilli The finding of the bacteria of these groups in pathological conditions affecting the alimentary tract has no ætiological significance

9 URINE

In any suspected case of bacterial infection of the urinary tract, examination of the urine should be made In cases of obstruction due to any cause such as calculus enlargement of the prostate or urethral stricture infection is liable to occur although the symptoms of such infection may be masked by those of obstruction.

Urine affords a suitable medium for the growth of many bacteria, hence it is essential to prevent contamination of the specimen by extraneous organisms and it is advisable that bacteriological examination should be carried out as soon as possible after the

urine has been collected In the male it is sufficient to wash thoroughly the glans penis and the meatus with 1 in 1 000 corrosive sublimate the urine is then passed into two sterile flasks the first of which is rejected in case contamination has occurred In the female after similar precautions as regards cleansing a sterile catheter must be used The bacteriologist should be notified whether or not the specimen is a catheter one and also of the date and hour of collection

If organisms are scanty the specimen should be centrifuged and films and cultures made from the sediment Films should be stained by Ziehl Neelsen s method for tubercle bacilli and by Gram s method for other bacteria In staining by Ziehl N elsen s method the film should be treated for one minute with alcohol after decolorizing with acid By this means the acid fast smegma bacillus which is frequently present on the external genitals and may contaminate the specimen is decolorized Tubercle bacilli in the urine occur either in small clumps or singly They are morphologically very characteristic but are often scanty In such cases better results will be obtained if the sediment of a twenty four hour sample is concentrated in the centrifuge and examined In cases of suspected renal tuberculosis where pus cells are present in the urine but no tubercle bacilli can be demonstrated microscop cally the inoculation of the urinary sediment into a guinea pig may provide evidence of tuberculous infection

In searching for other bacteria it is essential that the results of the examination of the Gram stained films should be amplified by culture the medium to be used being chosen in accordance with the findings in films The bacteria can then be identified by their cultural character fermentation reactions and if necessary serological tests

In some instances it may be desirable to ascertain the number of bacteria present in a sample of urine This can be conveniently done by plating known quantities of the fresh specimen (diluted if necessary) on a solid medium

Bacterial infection of the kidneys may be due to the tubercle bacillus *Staphylococcus aureus* or members of the colon typhoid paratyphoid group of bacteria Pyeliti is most frequently due to coliform bacilli while cystitis may be due to any of these bacteria or streptococci or possibly the gonococcus In cases of gonococcal urethritis gonococci are generally to be found in the urinary

sediment In undulant fever due to *Brucella abortus* or in Weil s disease the causative organisms may be found in the urine but as in the case of typhoid and paratyphoid infections their excretion may be confined to a certain stage of the disease or may be intermittent

10 PUS AND PURULENT EXUDATES

In the examination of pus and purulent exudates one should be guided in some degree by the clinical course of the infection the tissue or organ affected and as in the case of actinomycosis (*see* p 414) the naked-eye appearance of the fluid which may suggest a particular causal organism

Pus may be collected by means of a sterile syringe and should be sent to the laboratory in a suitably plugged tube Only in exceptional circumstances should a swab be used to collect the material

In general the examination of pus should be by films and culture Film preparations should be stained by Ziehl Neelsen s method if there is any possibility of tuberculous infection and by Gram s method for other organisms The preliminary examination of films will be a guide in the planting of cultures Cultures should in general be made on agar and blood agar If there is reason to suspect an anaerobic infection such as tetanus or gas gangrene a duplicate set of cultures on appropriate media will be set up under anaerobic conditions

The bacteria most commonly found in pus are staphylococci streptococci pneumococci and tubercle bacilli The gonococcus meningococcus coliform bacilli and actinomyces also cause suppurative lesions while pus formation may occasionally be associated with localized infections by bacteria which do not usually produce suppuration such as typhoid and paratyphoid bacilli *Brucella abortus* and *Hæmophilus influenzæ* In suppurative lesions following wounds and in those opening into a mucous surface more than one type of bacterium is frequently present

In gonorrhœa the ease with which the organism may be detected varies with the stage of the disease and the effect of treatment. In the urethral discharge of acute gonorrhœa in the male the appearance of Gram negative intracellular diplococci is sufficiently characteristic for diagnostic purposes In chronic cases where microscopical examination of the discharge or urinary sediment

is negative the organism may be grown on a suitable medium such as agar containing blood or serum Gonococci die out rapidly at room temperature and inoculated culture media should therefore be incubated at 37 C as soon as possible In the female adult patient material for examination should be obtained from the urethra or cervix—not from the vagina In cases of vulvo-vaginitis in children the vaginal discharge should be examined In chronic cases of gonorrhœa where microscopic and cultural methods have given negative results the complement fixation test may yield evidence of infection (*see* below)

Actinomycosis may be suspected if small yellow granules are present in the pus These granules can be readily seen if a few drops of pus are added to a tube of sterile saline or distilled water when they rapidly sink to the bottom If one of the granules is pressed between a slide and cover slip it will be seen to be composed of a central mass of filaments and homogeneous club shaped bodies may be seen at the periphery Films stained by Gram s method show Gram positive filaments of varying length some of which show branching The organism can be cultivated only under anaerobic conditions

In suspected cases of malignant pustule the anthrax bacillus may be demonstrated in films and cultures made with the exudate from the lesion or the fluid obtained from the blisters which are usually present

Serous exudates which have any considerable cellular content should be examined in the same way as pus If the cellular content is scanty centrifugation may be necessary as a preliminary

11 EXAMINATION FOR *SPIROCHÆTA PALLIDA*

In untreated syphilis in the primary stage careful examination of fluid from the chancre will usually reveal the causal spirochæte The sore should be cleaned with a sterile swab and saline and then is squeezed gently until a little serous fluid exudes *If the fluid* is blood stained this should be wiped away until a specimen free from blood can be obtained The fluid should be taken up in a piece of capillary glass tubing the ends of which are sealed with a flame and the specimen dispatched to the laboratory The fluid should be examined as a wet preparation under dark ground illumination The spirochæte is recognized by its size and close

spirals Where the equipment necessary for dark ground illumination is not available the spirochæte may be seen in films stained by Giemsa or Fontana s method but identification of the spirochæte in such films is not always easy In the less common extragenital chancres as for example on the lips or tongue the examination may be complicated by the fact that other spirochætes resembling the *Spirochæta pallida* may be present As the Wassermann reaction does not usually become positive until one to three weeks after the appearance of the primary sore the detection of the spirochæte is the most certain laboratory aid to diagnosis during this period

12 SERUM REACTIONS IN DIAGNOSIS

When a patient becomes infected with bacteria his tissues usually respond by the formation of antibodies Anti bodies are usually specific for the type of infecting bacterium and so the detection of antibodies against a specific organism may afford evidence as to the exact nature of the infection The evidence obtained by the examination of a patient s serum for antibodies can in most cases only be presumptive and is never of the same value as the detection of the infecting organism It is however of greater significance when a rising titre is demonstrated during the course of the illness Further as the production of specific antibodies is a response to the infection they will not usually appear during the first week or so of the disease so that negative serum tests at this time are no evidence against the presence of a suspected infection In general the infecting organism is most readily detected before the appearance of specific antibody The presence of antibody in a patient s serum may be revealed by the agglutination reaction or by the complement fixation test Blood is obtained by venepuncture as for culture except that the blood (5 0 ml.) is placed in a sterile tube and allowed to clot The serum is later separated from the clot and freed from blood cells by centrifugation if necessary

13 AGGLUTINATION REACTIONS

For these tests increasing dilutions of the patient s serum are mixed in small tubes with suspensions of the type of organisms with which the patient is believed to be infected The tubes are then placed in a water bath at 55 C for a few hours before being

examined The presence of specific agglutination will be indicated by the *appearance of clumps in the bacterial suspension* The agglutinating titre of the serum is the highest dilution which produces visible clumping of the bacteria

Standard suspensions of various organisms suitable for use in agglutination tests may be obtained from the Central Public Health Laboratories Colindale Avenue Colindale N W 9 (Tel Colindale 6041)

Agglutination tests with the patient s serum may be of value in the diagnosis of infections due to the typhoid and paratyphoid bacilli the dysentery bacilli *Brucella abortus* and the spirochæte of Weil s disease

In assessing the results of agglutination tests in suspected cases of enteric fever a previous history of prophylactic inoculation with typhoid vaccine or of enteric infection is important The agglutination titre of normal human serum for organisms of the typhoid paratyphoid group is low but a higher titre than normal may be present in individuals who have previously been inoculated with typhoid vaccine or suffered from typhoid fever The duration of illness is also important agglutinins in the serum are strong towards the end of the third week of enteric infection so that if a result of doubtful significance is obtained by the agglutination test in the second week of a patient s illness a second examination made a week later will be helpful

In bacillary dysentery the acute stage of the illness frequently lasts only a few days and a significant increase in serum agglutinins above normal does not occur in all cases A negative agglutination reaction against the various types of dysentery bacilli does *not therefore exclude the possibility of infection* with one of the types

In suspected cases of undulant fever the agglutination test is of great value as most cases of infection with *Brucella abortus show a high titre of agglutinins after the first week* of the disease

In testing for agglutinins against the spirochæte of Weil s disease dilutions of the serum are mixed with a living culture of the organism and the mixtures are examined microscopically by dark ground illumination *A positive reaction is indicated by the cessation of* motility and clumping of the organisms Practically all cases of Weil s disease give a well marked positive reaction after the first week of illness

14 COMPLEMENT FIXATION TESTS

These tests are based on the fact that serum containing antibody when mixed with a suitable antigen has the power of fixing a measurable amount of complement Serum to be tested is heated for 30 minutes at 56 C to destroy its own complement. In doing the test a known dose of complement is allowed to act for a given time with a known amount of antigen and the serum to be tested At the end of this time the presence or absence of free complement is tested for by adding a known amount of red cells which have been sensitized with a hæmolytic antibody If free complement is still present the red cells are laked and the reaction is negative If the complement has been fixed no hæmolysis takes place and the reaction is positive

The complement fixation test in the diagnosis of syphilis is usually known as the Wassermann reaction A positive reaction may be expected from five to seven weeks from the date of infection or from one to three weeks after the appearance of a primary sore The reaction is positive in all cases of secondary syphilis and practically all cases of untreated tertiary lesions A positive reaction will be obtained with the cerebro-spinal fluid in all cases of general paralysis in most cases of cerebral syphilis and less constantly in tabes dorsalis It will be seen therefore that although a negative reaction does not exclude a diagnosis of syphilis the reliability of the test is such that in the presence of a negative test strong evidence will be necessary to uphold such a diagnosis Remember that a positive reaction may be present in latent cases and therefore may be met with in patients suffering from some other disease

In the complement fixation test as applied in the diagnosis of gonococcal infection a suspension or extract of gonococci is used as antigen A positive reaction is not to be expected in the acute stage of gonorrhœa and may never be obtained in simple cases of urethritis The reaction is generally positive in cases with complications and is of especial value in chronic pelvic disease and in cases of arthritis and tenosynovitis

In cases of whooping-cough the serum obtained in the third week of the disease and later usually fixes complement in the presence of an antigen made from a suspension of *Hæmophilus pertussis* so that the test is most useful at that stage of the disease when cultural methods are likely to fail

In cases of hydatid disease the serum usually gives a positive complement fixation test in the presence of a hydatid antigen The reaction may therefore be of value in suspected cases

15 THE PAUL-BUNNELL TEST

The patient s serum is inactivated at 56 C for half an hour Dilutions are made with normal saline from 1 in 2 to 1 in 128 An equal volume of 2 per cent washed sheep cells is added giving *final dilutions of 1 in 4 to 1 in 256 The tubes are left over night* at room temperature and next morning each tube is gently inverted to look for agglutination A positive result is one in which *agglutination occurs in dilutions from 1 in 32 onwards* and may be found in glandular fever (infectious mononucleosis) and in serum sickness Positive reactions due to the latter condition may *be distinguished by the fact that this antibody* can be adsorbed out of the serum by minced guinea pig kidney

16 *VIRUS DISEASES*

The laboratory may help in the diagnosis of certain virus diseases by (1) the detection of a particular virus or (2) by determining the presence of specific antibodies in a patient s serum Success in detection will depend on whether the virus to be sought is capable of infecting laboratory animals or can be grown by the special methods used for the cultivation of viruses in living tissues *in vitro* The success of the second procedure will depend on whether a suitable antigen is available These conditions and the fact that the execution of such tests requires a specialized knowledge has imposed a limit on their use in routine laboratory diagnosis Labor atory investigations along the lines indicated may be useful in the following diseases

Psittacosis —Where the history and clinical picture suggests this disease sputum may be examined for the presence of virus After the first week the blood serum of the majority of cases *gives a* positive complement fixation test with a psittacosis antigen

Lymphocytic choriomeningitis —In the acute stage of this condi tion the virus may be isolated from the *cerebro spinal fluid* by inoculation of animals and in convalescence the blood serum may be examined for the presence of specific antibodies

Lymphogranuloma inguinale—Virus may be demonstrated in smears from the bubo by special staining methods or by inoculation of animals A skin test is also of value in later stages of the disease (*see* p 421)

Smallpox—In doubtful or atypical cases of smallpox the nature of the infection may be established by sending crusts of the lesions to a suitable laboratory Complement fixation tests with appropriate sera using extracts of the crusts as antigen may help in diagnosis

17 SKIN TESTS

Various skin tests are used as a measure of susceptibility of an individual s tissues to the test substances This susceptibility is indicated by an inflammatory reaction at the point of application or injection of the test substance it may indicate a normal susceptibility to toxic material e g bacterial toxin as in the Schick or Dick tests or it may indicate a state of hypersensitiveness or allergy to a bacterial or other protein as in the tuberculin test In the first instance a positive reaction may be expected in persons who have had no previous contact with the toxic substance whereas the second (allergic) type of response may be the result of sensitization through previous experience of the test substance

18 THE SCHICK TEST

This is a test of susceptibility or immunity to diphtheria toxin The toxin is so diluted that the test dose is contained in 0 2 ml and similarly diluted heated toxin is used as control Glass syringes graduated in tenths of a millilitre should be sterilized by boiling and allowed to dry before use a No 18 or 20 needle with short bevel should be used The skin of both forearms should be washed with soap and swabbed with alcohol or ether when dry the skin is stretched by holding the forearm firmly with the left hand the injection being made with the right Introduce the needle almost parallel with the skin and bevel upwards *when the bevel has disappeared inject* 0 2 ml The control heated toxin is injected into one forearm and the unheated toxin into the other In individuals who are not immune to the toxin a positive reaction will usually appear within 24 hours and will reach its

maximum on the 4th or 5th day Any non specific reaction due to the protein in the medium from which the toxin was prepared will show within 24 hours on the control arm but will usually have disappeared by the 4th day when the final reading should be made By this test it is possible to divide people into two groups immune and susceptible By inoculating the susceptibles with toxoid antitoxin mixtures or floccules it is possible to render them immune Where this practice has been widely adopted in young children it has been found possible to reduce the incidence of diphtheria to negligible proportions

19 THE DICK TEST

This test is similar to the Schick test but applies to scarlet fever and the toxin used is derived from scarlatinal streptococci The test is of doubtful value because of the difficulty of immunizing against streptococcal infection

20 THE TUBERCULIN TEST

This test is based on the fact that the tissues of a person infected with the tubercle bacillus become sensitized to tuberculin The older cutaneous test of Von Pirquet has been largely replaced by the more delicate intracutaneous method of Mantoux and the recently introduced patch test

Von Pirquet s cutaneous test —This test consists of placing a drop of Old Tuberculin on the forearm and a drop of 50 per cent glycerin broth adjacent to it as a control with a small lancet or needle a scratch is made through each of the drops A positive reaction is denoted by the appearance within 24 to 48 hours of a bright red papule at least 5 mm in diameter at the site of scarification through the tuberculin

The Mantoux test —In applying this test 0 1 ml of a 1 in 10 000 dilution of Old Tuberculin is injected intradermally into the skin of one forearm a similar dilution of glycerin broth being injected from another syringe into the other arm as control A positive reaction is characterized by the development within 48 to

72 hours of an area of erythema and infiltration 10 mm or more in diameter at the site of the injection of the tuberculin If the reaction is negative after 48 hours a dilution of 1 in 1 000 may then be injected and if this is negative a further test with 1 in 100 dilution may be made

The Patch test method —This method consists in applying to the skin by means of adhesive plaster tuberculin in the form of a powder or paste The necessary preparations have recently been placed on the market by various firms The skin is thoroughly cleansed with alcohol and ether and when dry the plaster bearing the tuberculin is applied The material is removed after 48 hours when the result is read

The value of the tuberculin test is limited owing to the fact that as a person gets older the less likely is he or she to have escaped previous clinical or sub-clinical tuberculous infection At the age of 1 year approximately 5 per cent at 5 years 20 per cent at 10 years 40 per cent at 15 years 60 per cent. and at 20 years or over about 90 per cent of individuals in an urban population give a positive reaction to tuberculin. In rural communities the percentage of positive reactions is rather less In young children a positive reaction will have greater significance while at all ages a negative reaction will be of value as evidence against a diagnosis of tuberculosis It must be remembered however that in the earliest stages of the disease the reaction may be negative and in very acute cases or in the last stages when no response from the tissues can be expected a negative reaction may be obtained

21 THE FREI TEST

This test is widely used in the diagnosis of lymphogranuloma inguinale The antigen consists of pus from a bubo or brain suspension from an experimentally infected mouse heated to 60 C for 2 hours Intracutaneous injection of 0 1 ml gives rise in an infected subject to an infiltrated inflammatory area at least 5 mm in diameter with a central zone of necrosis The reaction reaches its height in 48 or 72 hours

22 THE CASONI TEST

This test is useful in suspected hydatid disease The antigen is prepared from the fluid or walls of cysts and on injection intra dermally gives rise in positive cases to a reaction of the wheal and erythema type *The reaction appears in 5 or 10 minutes and* reaches its maximum within about an hour

23 TESTS FOR SENSITIVITY TO VARIOUS ANIMAL OR VEGETABLE PROTEINS

In the investigation of patients suffering from allergic states such as asthma or hay fever the sensitiveness to various animal or plant proteins may be determined by skin tests Sterile watery extracts of such materials as horse hair feathers various pollens fish muscle fruit eggs etc are on the market in a form ready for use A single line of scarification about 1 cm long is made through a drop of fluid and various solutions may be tested at one time Sensitivity to a particular extract is indicated by a reaction of the wheal and erythema type which appears within 10 minutes and attains its greatest size in from 30 minutes to an hour The examination of the responses to these skin tests in a patient suffering from hay fever or asthma may provide information of value in the prevention of further attacks

24 PREGNANCY TESTS

The urine of both normal men and women contains small amounts of the gonad stimulating hormone prolan derived from the anterior lobe of the pituitary During pregnancy prolan is also produced by the chorionic cells of the placenta and the amount in the urine is increased It is greatly increased in certain abnormal conditions such as hydatidiform mole chorionearcinoma and teratoma of the testis This is the basis of the Ascheim Zondek and Friedmann tests Urine is injected into immature female test animals and its prolan content is estimated by observing the effects of the injections on the animals ovaries About 30 ml of urine are required A concentrated early morning specimen should be obtained and a few crystals of boric acid should be added as a preservative

In the Ascheim Zondek test four or five immature white mice

each receive six subcutaneou injections of 0 5 ml of urine in three days The ovaries are then dissected out and examined In a positive test the uteri are enlarged and hæmorrhagic follicles or corpora lutea are found in the ovaries This te t may be done quantitatively by using graded dilutions of urine In normal pregnancy it is unusual to obtain a positive test in dilutions greater than 1 in 50 With hydatidiform mole or chorioncarcinoma, positive tests with dilutions up to 1 in 1 000 or more may be found The Ascheim Zondek test becomes positive about three weeks after the first missed period With proper precaution and tech nique it has a high degree of accuracy

The Friedmann test becomes positive even earlier in pregnancy and can give a result in 36 hours One or better two immature female rabbits are given two intravenous injections of 10 ml of urine at twelve hour intervals and are killed and examined as described above twenty four hours after the second injection The results of this test are less reliable than those of the Ascheim Zondek

APPENDIX

WEIGHTS AND MEASURES

1 Imperial weights and measures

1 grain gr	
1 ounce oz	= 437 5 grains
1 pound lb	= 16 ounces = 7 000 grains
1 minim	= 0 91146 grain
1 fluid drachm	= 60 minims
1 fluid ounce	= 8 fluid drachms
1 pint	= 20 fluid ounces
1 gallon	= 8 pints

2 Relation between Imperial and metric systems

1 grain	= 64 8 milligrammes (mg)
1 ounce	= 28 3 grammes (grm)
1 lb	= 453 6 grammes
1 gramme	= 15 432 grains.
1 kilo	= 2 lb 3 oz
1 minim	= 0 059 millilitres (ml)
1 fluid drachm	3 5 ml
1 fluid ounce	= 28 39 ml
1 pint	= 567 9 ml
1 ml (c c)	16 9 minims
1 litre	= 35 2 fluid ounces
1 inch	= 2 54 centimetres (cm)
1 foot	= 30 48 cm
1 yard	= 91 44 cm
1 cm.	= 0 39 in
1 metre	= 39 37 in

3 Conversions

To convert grammes per 100 ml into grains per ounce multiply by 4 375

To convert grammes into ounces avoirdupois multiply by 10 and divide by 283

To convert litres into pints multiply by 88 and divide by 50

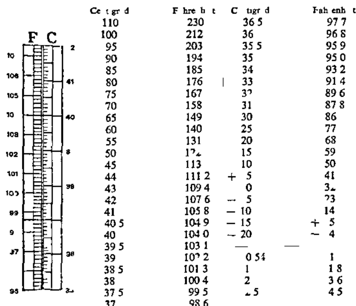
To convert kilos into pounds multiply by 1 000 and divide by 454

4 Centigrade and Fahrenheit scales

To convert Fahrenheit into Centigrade subtract 32 multiply the remainder by 5 and divide the result by 9

To convert Centigrade into Fahrenheit multiply by 9 divide by 5 and add 32

The following table and figures show the relation of degrees Fahrenheit to Centigrade as far as is likely to be required in clinical work —



Centigrade	Fahrenheit	Centigrade	Fahrenheit
110	230	36 5	97 7
100	212	36	96 8
95	203	35 5	95 9
90	194	35	95 0
85	185	34	93 2
80	176	33	91 4
75	167	32	89 6
70	158	31	87 8
65	149	30	86
60	140	25	77
55	131	20	68
50	122	15	59
45	113	10	50
44	111 2	+ 5	41
43	109 4	0	32
42	107 6	− 5	23
41	105 8	− 10	14
40 5	104 9	− 15	+ 5
40	104 0	− 20	− 4
39 5	103 1	—	—
39	102 2	0 54	1
38 5	101 3	1	1 8
38	100 4	2	3 6
37 5	99 5	2 5	4 5
37	98 6		

Fig 96
Fahrenheit and Centigrade scales compared

5 Congo red test papers

These are made by soaking filter paper in a solution of Congo red of the strength of 1 decigramme to 100 ml of water or in a saturated alcoholic solution They are allowed to dry and are then ready for use

SOLUTIONS REQUIRED FOR URINARY TESTING

6 Hypobromite solution

Dissolve 100 grm of caustic soda in 250 ml of water cool then add 25 ml of bromine The solution is apt to undergo the following decomposition



$$3NaBrO = 2NaBr + NaBrO_3$$

It is therefore better to prepare it as required by adding 2 5 ml of bromine to 25 ml of the caustic soda solution

The bromine is supplied in small tubes which readily break when shaken up smartly with the soda solution in a stout stoppered bottle

7 Esbach s reagent

Dissolve 10 grm of picric acid and 20 grm of citric acid in about 900 ml of boiling water cool, and add water to 1 litre

8 Fehling s solution

(*a*) Take 34 64 grm of powdered crystalline copper sulphate dissolve in 200 ml of warm distilled water cool and fill up to 500 ml

(*b*) Dissolve 180 grm of crystallized sodium potassium tartrate in 300 ml of hot water filter and add 70 grm of pure caustic soda or 100 grm of potash cool fill up to 500 ml

When required mix equal volumes of (*a*) and (*b*) The result is an alkaline solution of potassic cupric tartrate of which 1 ml is exactly reduced by 5 mg of pure glucose

9 Benedict s reagent

Copper sulphate	17 3 grm
Sodium citrate	173 grm
Anhydrous sodium carbonate	100 grm
(or crystalline sodium carbonate	200 grm)

Dissolve the copper sulphate in 100–150 ml of distilled water and add slowly with constant stirring the other ingredients dissolved in distilled water and filtered this should amount to about 800 ml Finally make up to 1 litre with distilled water

SOLUTIONS REQUIRED IN THE EXAMINATION OF BLOOD

10 Hayem s solution (for counting red cells)

Sodium chloride	1 grm
Sulphate of soda	5 grm
Corrosive sublimate	0 5 grm
Distilled water	200 ml

11 Diluting fluid for hæmocytometer (for counting white cells)

Gentian violet (1 per cent)	0 1 ml
Acetic acid	2 ml
Distilled water	100 ml

12 Toisson s solution has the following formula

Methyl violet 5B	0 025 grm
Sod chlor	1 000 grm
Sod sulph	8 000 grm
Neut glycerin	30 ml
Aq destill	160 ml

Should be filtered just before use

SOME STAINING METHODS

13 Gram s method (Lillie s modification)

The original Gram s method involves the use of aniline water gentian violet This is an unstable solution and requires to be made up fresh at frequent intervals It is recommended therefore that Lillie s modification should be used

To stain by Lillie s modification of Gram s method —

(1) Stain for one minute with ammonium oxalate crystal violet solution (2 grm crystal violet 20 ml absolute alcohol 80 mL of 1 per cent aqueous solution of ammonium oxalate)

(2) Pour off the crystal violet solution and pour on strong Lugol s solution (iodine 1 part potassium iodide 2 parts distilled water 100 parts) Pour off and add a fresh quantity of iodine solution Leave for half a minute

(3) Wash in methylated spirit and continue until the violet colour ceases to run from the film.

(4) Counterstain with carbol fuchsin diluted 1 in 10 with tap water

(5) Wash with water and blot dry

14 Ziehl Neelson s method of staining for tubercle bacilli

(1) Carbol fuchsin

Basic fuchsin	1 grm
Absolute alcohol	10 ml
5 per cent aqueous carbolic acid	90 mL

(2) 25 per cent aqueous solution of sulphuric acid

(3) Loeffler s methylene blue

Saturated alcoholic solution of methylene blue	30 ml
Distilled water	100 ml
1 per cent solution of caustic potash	1 ml

Pour on boilin carbol fuchsin and leave for 7 minutes Wash off and place in the acid until on washing in water not more than a faint pink colour returns Pour on the methylene blue solution and leave for one minute Wash in water blot and dry

15 Loeffler s stain (This should be freshly prepared)

Concentrated alcoholic solution of methylene blue	1 ml
Caustic potash in 0 1 per cent aqueous solution	3 ml

Specimens are stained in 5 to 30 minutes Excess of stain is discharged by rapid washing in water acidulated with acetic acid (2 drops of acid in a small bowl of water) and all traces of acid are well washed out The specimen i then dried and mounted

16 Carbol thionin blue (Prepared freshly)

Saturated solution of thionin blue in 50 per cent alcohol	10 ml
1 in 40 solution of phenol in water	100 ml

This stain is one of the best for film preparations After staining which is rapidly effected wash the specimen in water then dry and mount Sections should after washing be passed through alcohol containing a trace of ammonia thereafter dehydrated by absolute alcohol cleared with xylol and mounted in balsam

17 Neisser s method of staining B diphtheriæ

Stain No I	Methylene blue	1 grm
	Absolute alcohol	20 ml
	Glacial acetic acid	50 ml
	Distilled water	1 000 ml
Stain No II	Crystal violet	1 grm
	Absolute alcohol	10 grm
	Distilled water	300 ml
Stain No III	Chrysoidin	1 grm
	Distilled water	300 ml
	Heat to dissolve and filter	

The film should be fixed by heat

Two parts of No I stain should be added to one part of No II Stain with this mixture for 5 minutes Wash with water and then stain with No III for 10 seconds Wash in water and blot

The mixture of I and II must be made up fresh daily

18 Hiss s method of capsule staining

This is very useful for the demonstration of pneumococci in the sputum or in pneumococcal exudates The stain consists of 1 part of a saturated

alcoholic solution of fuchsin and 19 parts of distilled water A film of the material to be examined having been dried and fixed has a few drops of the stain placed upon it and is heated for a few seconds until steam rises The stain is then washed off with a 0 per cent solution of copper sulphate without being washed in water the preparation is dried between filter papers and mounted in balsam.

19 The Romanowsky stains

These stains depend for their action on the compounds formed by the interaction of methylene blue and eosin as originally introduced by Romanowsky They are used for staining blood films the cells in pathological fluids films or tissues containing bacteria and material containing protozoal parasites e g malaria

The chief combinations in use are those of Leishman Jenner Giemsa, and J H Wright Powder for the preparation of these stains can be obtained from Messrs Baird and Tatlock or Soloids can be had from Burroughs Well ome & Co through any dealer

Leishman s preparation is perhaps that most used and is prepared by dissolving one soloid in 10 ml of *pure* methyl alcohol It is of great importance that the methyl alcohol should be acetone free

The instructions sent with the various soloids should be strictly followed, and the bottle in which the stain is stored must be well stoppered If the preparation is too blue this may be corrected by careful washing with acetic acid, 1 in 1 500 if the eosin tint is too strong it can be lightened by the use of 1 in 7 000 solution of caustic soda The appli ation of the stain is described in Chap V (p 147)

With this stain the red blood-corpuscles are coloured pink the nuclei of leucocytes a reddish purple and any acidophil or basophil granules pink and blue respectively The nu lear substance of the malarial para site is stained a reddish purple In trypanosomes the two nuclei have a purplish stain the protoplasm is blue and the edge of the undulatory membrane is stained pink

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